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## ORIGINAL ARTICLES

### MY EXPERIENCE WITH CERTAIN INDUSTRIAL POISONS*

BY WILLIAM F. BOOS, M.D.

I AM frequently called upon for advice in relation to both the diagnosis and the treatment of chemical and medical injuries sustained in a variety of industrial occupations. My work does not concern itself in any way with injuries caused by surgical traumatism.

In many of the cases that are referred to me, the cause of the injury is not definitely known; in others, certain possible causes are alleged, but the clinical picture is not sufficiently like that which has been seen in the past to warrant a definite diagnosis. Another class of cases that I am asked to see includes chemical and medical injuries which have not responded properly to the treatment used by the plant physician or the doctor of the insurance company.

It has been brought home to me very forcibly that in both categories of cases the lack of chemical training on the part of the physicians who see the cases first, or who treat them subsequently, is the main cause of failure to obtain satisfactory results. In other words, the recognition of a chemical injury and the subsequent treatment of such an injury require, first of all, a knowledge of the chemical behavior of the agent producing the injury. I realize, of course, that it is not feasible to obtain, for plant work, physicians who are well-rounded chemists; still, the kind of chemical injuries which result in a given industrial plant are usually so limited in scope that it would not be difficult to teach the physician in charge the chemistry of the few chemical agents with which he has to contend. But in a true chemical manufacturing plant which puts out a variety of corrosive and poisonous products, the physician should, I think, be really well trained in chemistry, and I believe that in this branch of industry the physician of the future will be primarily a chemist.

However, the queston of the greatest importance at the moment is: Can we get satisfactory results with the physicians now available, and how? I think this question may be answered in the affirmative, because, to my mind, it is merely a matter of intelligent coöperation between the plant physician and the chemical expert of the plant.

In every plant of any importance which manufactures or uses corrosive or poisonous chemical agents, there is some person who has an expert knowledge of the chemical behavior of the reagents which are made or used; he is not always called a chemist, however; sometimes he is the engineer, or he may be the superintendent himself. This official—whoever he may be—can always give the doctor valuable advice, because he knows which reagents to use to neutralize, precipitate, or in other ways render harmless the agent producing the injury.

Let me give you an example: Several years ago I was asked to inspect a chrome-tanning plant in which a number of employes had suffered repeated and serious chrome burns of the hands, arms, face, and even of the abdomen. When a man complained of chrome burns, it was the practice to send him to the plant physician for treatment; and the doctor, as is usual with practitioners, applied boric ointment, bandaged the part, and sent the man home with instructions to report daily for renewal of the dressing.

What was the result? The lesions became more and more serious, and after a week or so the employe's injury was in a most deplorable state. This is what happens: The chrome acid which was spattered on, let us say, the man's arms, penetrates very slowly through the skin, and does not at first produce symptoms. But after about twenty-four hours the employe begins to feel a sensation of itching and burning which grows steadily worse. The doctor then applies an ointment, and bandages the arm. By so doing he effectually prevents the removal of the chromic acid; the latter continues to penetrate into the lower layers of the skin, and, at the end of a week the employe is suffering agonies from multiple, deep chrome burns.

The men who are subject to these burns are employes who take the skins from the chrome bath (a mixture of dilute hydrochloric acid and bichromate of potash), and place them first in the washing machine and later in the "hypo" bath. I asked the superintendent who accompanied me if the men who handled the skins after they had been through the hypo bath were ever affected by chrome burns, and he answered: "Of course not." His answer was evi-

*Read at the 14th Annual Congress of the National Safety Council, October 1, 1925, Cleveland, Ohio.

dence that he knew that the hypo solution immediately neutralizes the chromic acid, rendering it harmless—but did he ever tell the doctor about it? No! And there, in the middle of the floor, was standing a great vat of hypo solution, the most efficient cure for, and preventive of, chrome burns known. But because chrome burns were thought to be a *medical* injury or a *disease*, as it were, the superintendent never dreamed to use his common sense.

That is where the trouble lies in most cases. The chemist or engineer is afraid to handle a case which he thinks is medical, while the doctor attempts to cure a chemical injury without having the slightest knowledge how to neutralize the injurious agent, or otherwise to stop its action on the tissues. But if, for example, the doctor were to say to the engineer: "Mr. X., I have a man here with some chrome burns on his arms; how can I make that stuff harmless?" the engineer would immediately suggest the use of the hypo solution, and the burns would be nipped in the bud.

A similar consultation in other cases of chemical injury would produce equally effective results in safeguarding the health of the employes, and, incidentally, it would save the insurance company much compensation money.

In order to emphasize the importance of coöperation between doctor and chemist, or engineer, I am going to tell you about some short cuts in both the diagnosis and the treatment of a variety of industrial poisonings which were referred to me mainly because of the lack of such coöperation. It will be best for me to classify the active chemical agents to some extent, according to their chemical and physical properties.

### NITROUS FUMES

During my service as physician in chief of a munitions plant which was operated during the World War, I became very famliar with nitrous fume poisoning. There had been, at this plant, a number of fatal cases from the inhalation of nitrous fumes, and it was mainly to correct this evil that I was placed in charge of the medical work at the plant.

In this short paper I can give you merely an outline of this interesting and dangerous form of poisoning. Any of you who may desire to know more about the subject will be able to obtain detailed information from a special paper dealing with nitrous-fume poisoning, which I have in preparation, and which will be published shortly.

The active principle of nitrous fumes is $NO_2$, an acid gas. When it is first inhaled, this gas causes coughing, a choking sensation, pain in the chest, and the expectoration of yellow-tinged sputum. If the exposure ceases, the symptoms subside, and the employe who has been exposed to the fumes often feels strong enough to walk several miles to his home. Then, suddenly, he exhibits fulminating symptoms of respiratory disease with progressive edema of the lungs, and in a few hours he is dead. Some cases recover from the initial edema only to develop pneumonia within twenty-four to thirty-six hours. Many of the pneumonia cases die, some recover. After the edema sets in there is almost nothing one can do in the way of treatment, and the outcome is simply a question of individual resistance. But if the condition is recognized at the start and chemical common sense is applied, the victim of nitrous fume poisoning can be saved in every instance.

The present medical treatment for nitrous-fume poisoning as given in books on the subject, i. e., the inhalator and oxygen, is absolutely futile. What is needed is something to stop at once the action of the nitric and nitrous acids which are formed on the mucous membrane of the respiratory tract, and there is only one agent that will do this; namely, ammonia gas. Use an inhalator if you will, but be sure to have the air which is taken into the lungs bubble through a dilute solution of ammonium hydrate. However, the simplest and best method is to hold a pepper shaker filled with lumps of ammonium carbonate to the victim's nose, and make him deeply inhale the escaping ammonia gas. The latter immediately neutralizes the acids with the formation of harmless ammonium nitrate and nitrite.

The same principle applies, of course, to most acid gases and vapors, such, for instance, as chlorine, bromine, iodine and sulphur dioxide. It should not be used however, with hydrocyanic acid, which poisons not through any irritant acid properties, but by reason of a specific action of the CN ion on the respiratory center, causing paralysis of respiration with consequent asphyxia. As long as the victim of HCN poisoning is still breathing there is hope of saving him by artificial respiration, because hydrocyanic acid, being very volatile, is quickly excreted.

### BENZOL

In the case of benzol poisoning there is no chemical antidote, and the only measures which give any hope of succor are immediate removal to fresh air and artificial respiration. In one case which came under my observation a man had been ordered to clean the inside of a benzol retort which had but one tubulation. He was heard to laugh and shout boisterously. A fellow worker who recognized in these symptoms the beginnings of benzol intoxication tried to pull him out. Failing in this, he crawled into the retort himself, and with much effort succeeded in pushing his already unconscious friend out into the open. But the time required was too long, and the rescuer died in the retort, while the first victim recovered.

### TURPENTINE

Turpentine poisoning, again, is an important form of industrial poisoning, because the usual medical diagnosis is lead poisoning. As an example of this common error, I will cite two cases which were referred to me at the same time from a plant manufacturing automobile bodies.

These men were employed to spray a black varnish or finish on the bodies. They were gradually taken ill with extreme pallor, nausea, vomiting, abdominal cramps, and a form of neuritis. The local doctor saw them, and because they were said to be painters and on account of the symptoms given above—all of which are in keeping with lead poisoning—he called them lead cases. They were referred to me for treatment.

Neither of the men showed a lead line; there was no suggestion of wrist or toe drop, and the excreta did not contain lead. The red corpuscles showed granular stippling associated, however, with a low percentage of hemoglobin. I asked for samples of all the materials used by the men in spraying and found them lead-free. Further study of the cases then convinced me that they were cases of turpentine poisoning.

A suggestive fact in these cases was the statement, on the part of both men, that they never had felt any ill effects during the summer, but that their symptoms had started with the closing of the shop windows in the cold weather.

I think it is very evident that these cases were not properly investigated before the diagnosis was made, because, as I learned subsequently, the superintendent knew perfectly well that the spraying material was lead-free and it was a puzzle to him how the men got their lead. Nevertheless, he accepted the medical diagnosis and did not discuss the matter with the doctor. Perhaps he thought that some volatile compound of lead emanating from another part of the factory was the cause of their illness; for there seems to be a more or less generalized belief that such compounds do occur in the ordinary lead and paint industries. This, of course, is absolutely erroneous; when lead posoning comes through the respiratory tract, it is some form of lead dust that is inhaled.

But to return for a moment to turpentine—I shall have occasion later to say a little more about plumbism—I have seen so many cases of disability caused by turpentine, all of which were diagnosed as lead poisoning, that I have decided to publish my material, giving special attention to the differential diagnosis between the two forms of intoxication.

### WOOD ALCOHOL

Wood-alcohol vapors frequently cause pronounced irritation of the mucous membranes of the eyes and nose. This alcohol is used by the optical trades in conjunction with other substances, such as banana oil, in special cleansing fluids. The cases which I have seen were called poisoning by banana oil; the fumes of the latter are, however, quite harmless, and when grain alcohol—or grain alcohol denatured according to some formula which does not call for wood alcohol—is used in place of wood alcohol, the irritant properties of the cleanser disappear.

### ACID AND ALKALI BURNS

It is in cases of acid and alkali burns that a lack of chemical sense is particularly serious. Many such burns have been referred to me because they grew steadily worse under the treatmnt of the plant or local physician. The reason for this will be apparent when I tell you that for the average doctor a burn is a burn no matter what the cause, and the treament is always the same, namely, carron oil (equal parts of linseed oil and lime water) or boric ointment. These two preparations make an excellent protective coating against the removal of acid, alkali, or, in general, corrosive poisons, and as the result of their application the destructive penetration of the skin continues, until, at times, the loss of a limb is threatened.

When a chemical burn is sent to the doctor, he must find out what caused the burn, so that he can immediately render the corrosive agent harmless, either by neutralization or by precipitation in form of an insoluble salt. Now, questioning of foreign, ignorant laborers does not lead far; they often work with both acids and alkalies, and they don't even know the names of any of the reagents. As a rule, too, the foreman doesn't know what caused the burn, or he may be entirely mistaken in his opinion, and may thus cause the doctor to err through misinformation. Therefore—in the vernacular—it is up to the doctor to find out for himself, and he can do this by applying bits of moist litmus paper, both blue and red, directly to the wound. If the paper turns red, he bathes—or better—soaks, the inured part in a five-per cent solution of bicarbonate of soda until all trace of the acid has disappeared. If, on the other hand, the paper turns blue, he employs a three-per cent solution of acetic acid as a wash or bath. Then, when he feels sure that the neutralizing wash has thoroughly penetrated the wound, he may resort to ointment and bandage.

Alkali wounds are more apt to give trouble than acid wounds, because the natural alkalinity of the tissues tends to neutralize the action of an acid agent; then, too, the acid albuminate which is formed is difficulty soluble and, in time, checks the further progress of the acid into the tissues; while alkalies, on the other hand, form very soluble alkali albuminates, which do not interfere with the continued pene-

trating action of the alkali into the defenceless tissues. For this reason alkali wounds must be bathed a very long time, and preferably with occasional change of the dilute acetic acid. When the latter is not available, equal parts of vinegar and water will do just as well.

### CHROME BURNS

I have spoken previously of chrome burns. When these are still small and nodular, or when the vesicular stage is just beginning, the bath of hypo solution (sodium thiosulphate) is the best neutralizer. But in the later stages, when the burn is deep, painful, and very tender, it should be treated with U. S. P. lead and opium wash, the lead-acetate content of which causes insoluble and harmless chromate of lead to be formed, while the opium soothes the pain. Look out for chrome burns! I don't know any caustic agent that will keep on burrowing and burrowing into the tissues like chromic acid. I once saw a hole in the back of a poor fellow's hand which went almost through to the palm, and yet it was just a neglected chrome burn.

Polishers and finishers of leather shoes are frequently the victims of a very persistent form of dermatitis which resembles eczema. The irritation is commonly due to alkaline agents which are present in the finish or dressing, one that is used for patent-leather shoes being the greatest offender.

It is almost impossible to compel the employe to wear gloves at his work, but it would be feasible to place within his reach a dilute solution of acetic acid to be used occasionally as a wash for his hands.

Oxalic acid is often present in dressings and bleaching fluids for white shoes. It produces a dry, scaly dermatitis of a very persistent character. Oxalic acid is practically as injurious for canvas and leather, as it is injurious for the human skin, and in my opinion its use should be discontinued.

### CYANIDE RASH

In the jewelry and watch making trade and in silver plating, cyanide solutions are much used. They cause localized burns, but more frequently they produce an eczema-like eruption of the skin of the hands, arms, and even of the face. The involvement of the face is due to thoughtless scratching or rubbing of the chin, nose, forehead or eyebrows by a worker whose hands have been in the solution; women especially, have a way of brushing back a lock of hair from the temple or forehead while they are dipping pieces of jewelry into a cyanide solution, and these are therefore favorite sites of the cyanide rash with female workers. Frequent repetition of such transference to the skin of the face is apt to produce a very resistant dermatitis. In these cases, again, the local physician diagnoses eczema, and treats the lesions with an alkaline wash which, of course, tends to intensify the cyanide action. As in all cases of alkali burns, the affected parts should at once be treated with a dilute solution of acetic acid to neutralize the cyanide, and thus to stop the alkaline penetration.

### PHENOL

Phenol burns are among the lesions which require very prompt attention; but there is nothing which can be used to neutralize the phenol itself, and on account of its slight solubility in water, washing of the affected part with plain water is simply a waste of time. Phenol is, however, very soluble in alcohol, and therefore this solvent can be used to advantage in removing the phenol from the injured part. In plants where carbolic acid is used to any extent, there should therefore be an abundance of first-aid cabinets containing large bottles of some form of denatured alcohol. One difficulty with this prophylactic measure lies in the craze on the part of many workers for all kinds of alcohol. The supply could be protected against pilfering, however, by having the door of the cabinet opened by an electric button connected with an alarm.

Phenol is taken up quite readily from the skin, and if a large area has been exposed to it, death may easily result from the action of the absorbed phenol on the central nervous system. In cases of this kind the local action on the skin may be very slight.

### HEAVY METALS

The heavy metal which interests us most is lead. Mercury and arsenic also claim their victims, but I see cases of poisoning by these two metals only very rarely. I mean, of course, industrial cases; of homicides and suicides from both mercury and arsenic I have seen my share but industrially speaking, arsenic and mercury poisoning are infrequent in the Boston district. I recollect, however, having seen several cases of chronic mercurial poisoning in thermometer makers.

The number of lead cases I have been asked to see is very large and I feel that the subject of lead poisoning is a most important one. Its recognition requires a very careful physical examination and complete examination of the blood, urine and stool as well. I do not intend, in this general paper, to dwell at any length on the subject of lead, but I would like to call attention to certain facts:

Lead compounds being quite easily absorbed through the mouth or the respiratory tract most of the employes who are brought into daily contact with them absorb more or less lead and, as a result, they are constantly excreting traces of the metal; this, of course, without necessarily showing any signs or symptoms of lead poisoning. In other words, the fact in itself that a man is excreting slight

traces of lead in the urine or stool, does not mean that he has lead poisoning; it is only when the complete clinical picture is strongly suggestive of lead poisoning that the presence of lead in the excreta is of importance. There is no question that many employes are being paid compensation insurance for illnesses that have nothing to do with their work, simply because lead was found in the excreta. A case in point:

L. H., 53, foreman in a paint shop. Has been a painter 35 years, but never before has had an attack of painter's colic. For the past year he has suffered from abdominal cramps, coming at first once a month, then every week, now every second day. No paralyses, questionable lead line on lower incisors, slight granular stippling of red cells, trace of lead in urine and stool.

My physical examination convinced me that his tenderness and colic were associated with the right kidney, and when I asked him to bring me the twenty-four hour urine, I found it to be dark red with blood. The man had a stone in the right kidney. It was removed by operation, and he made a complete recovery.

The lead in the excreta and the lead line, as also the granular stippling of a few red cells —the result of a profound secondary anaemia —completely misled his doctor. The employe in question is a man of careful habits, who never fails to wash his hands before eating a meal. Since he never scrapes down old paint, he is not exposed to the inhalation of lead dust, and he has been a painter for thirty-five years without ever having had an attack of painter's colic. These facts should have made the doctor hesitate in making a diagnosis of plumbism.

A word about the treatment of lead cases: The main organ of excretion is the liver, and therefore its activity should be stimulated by bile or bile salt medication, coupled with a good cathartic, such as licorice powder. As a result of this medication we get relief of constipation and increased elimination of lead from the system. The local paralyses should be treated by the air-cooled ultra-violet lamp in conjunction with massage. Potassium iodide may help to improve the general condition but as a lead eliminant it is a fallacy.

### COPPER AND ZINC

Industrial copper poisoning, I think, is unknown. A diagnosis of zinc poisoning should not be made in the absence of the characteristic and ever-present zinc (or brass) chills.

### TRADE ANAPHYLAXIS

A subject which has claimed the attention of physicians more recently, and which I think is destined to play a part of some importance in industrial medicine, is that of trade anaphylaxis.

It has been found that in a variety of trades certain workers develop a peculiar symptom-complex similar of that of hay fever, i. e., swelling and watering of the mucous membranes of the nose and eyes, sneezing, cough, bronchitis, and asthma. The reason for this illness is to be found in their sensitiveness to the proteids contained in the material with which they work. Thus we find millers and bakers sensitive to wheat- or rye-flour dust, and leather workers, who are troubled by the dust from some special kind of leather, be it cowhide, horsehide, pigskin, and the like. Some carpenters and cabinetmakers are made ill by the dust from certain kinds of wood, such as pine, oak, maple, birch, hemlock, mahogany, and so forth. Mattress makers are troubled at times by the horse dander in the horsehair which they use, and wool sorters are occasionally sensitive to the sheep's dander in raw wool.

Trade anaphylaxis should be thought of in all cases where the symptom-complex which I have described above is alleged to be the result of working in a dusty atmosphere, which, however, has not troubled other workers.

Before I close I shall say a few words about a form of industrial poisoning which is not chemical. I refer to the cases of furunculosis occurring in trades which require the use of oil for various kinds of lubrication. In some cases the skin injury results from an irritant in the oil itself, but I think these cases are rare. It has been my experience that the workers affected are usually uncleanly in their habits; they never bathe, and therefore oils, especially the heavier ones, act mechanically to occlude the pores in the skin, and to keep them plugged with dirty, infected material which favors the development of furuncles and boils. In all trades therefore in which the workers are apt to have their arms spattered or smeared with oil, it should be made obligatory for the employe thoroughly to scrub his arms and hands with a soft brush and much soap and water before he leaves the plant. This procedure, in my opinion, will eliminate most cases of furunculosis from oil or grease.

## A YEAR OF ENDOSCOPY

BY LYMAN RICHARDS, M.D.

ENDOSCOPY, or the inspection of certain body passages by electrically lighted instruments, has of late years become increasingly serviceable in the examination of the upper respiratory and alimentary tracts. With the progress in efficiency of the instruments and the technical skill in their use, laryngoscopy, bronchoscopy and esophagoscopy are now regarded as border-

line fields falling within the domain of the laryngologist, who is thus able to shed diagnostic and therapeutic light on structures that were formerly incapable of examination and treatment. The brilliant inventions of such pioneers as Brunings, Jackson and Mosher have made it possible for other men in various parts of the country to take up this work and to become more and more proficient in it. Among these are some few who find themselves able to devote their entire time to endoscopic procedures, being associated with clinics or hospitals to which cases of this sort are referred from widely distant points. Needless to say such men are at a great advantage and are consequently able to develop their diagnostic and operative skill to a far greater degree than those less fortunately situated who see such endoscopic cases only at rare intervals and whose lack of practice handicaps them in dealing with them. Nevertheless even these infrequent cases must be cared for since they cannot all be referred to the larger special clinics, and hence less experienced endoscopists are faced with the problem of carrying on their work under this disadvantage and of endeavoring to perform it as safely and as efficiently as possible.

It is with a view to presenting some phases of the work from this aspect that the author has collected all his endoscopic cases for the past twelve months in order to give some idea of their nature as they occur, not as a specialty, but as a part of general hospital work.

Wherever endoscopy is undertaken certain fundamentals must be observed if successful results are to be obtained. More than any other line of surgery, these procedures require a perfectly working and thoroughly adequate instrumental equipment which can be depended upon under all circumstances and in any emergency. The failure of any part of the working unit at a critical moment may be a matter of life or death. Another highly important essential is an operating team the members of which are familiar with the operator's needs and the fundamentals of the work itself. It is in this particular that the "occasional" endoscopist finds great difficulty since in the average hospital his assisting personnel is constantly changing and he is forced to train new assistants at frequent intervals.

Still more important for success is the development of technique and skill on the part of the operator himself. This can only be brought about efficiently by frequent practice in the laboratory and on the manikin. The latter in simple or complicated form is always available and furnishes a ready and in many ways very satisfactory means of acquiring the reflex movements which play so important a role in carrying out endoscopic procedures successfully.

It is not the purpose of this paper to discuss actual operative technique or individual methods of its execution. It is desired simply to present this work as it has been performed throughout the year, to show its range under ordinary conditions, the problems arising in connection with it, and some of the results obtained.

### LARYNGOSCOPIC GROUP

This group of cases comprises those in which the patients presented themselves for relief from some form of laryngeal obstruction, hoarseness, or aphonia. They were largely children, seen in the out-patient department, from which they were referred to the hospital for laryngoscopic examination. Many of the cases were of a similar nature so that only examples of particular diseases are recorded here.

The first case is representative of a small group of four infants who were examined for a question of laryngeal stridor. They all presented essentially the same picture and the appearance of the larynx in each was almost identical. There was an apparent redundancy of the mucous membrane covering the arytenoids to such an extent that the glottic lumen was encroached upon and simulated the wearing of an over large coat. None of these patients suffered from very serious dyspnea but all were stridulous and tended to have attacks of choking with mild cyanosis. No particular treatment is indicated other than careful directions to avert the results of a sudden laryngeal edema, and future return to the hospital for observation. Prognosis is considered generally good, the children tend to outgrow the condition, since the larynx apparently grows up to the temporarily redundant mucosa and finally the stridor disappears.

CASE I. "Boy, J. L., 4 weeks old. Full term baby, normal delivery. On mother's return from the hospital child exhibited difficulty in breathing and became blue when he cried. No history of foreign body.

"Child breathes with stridor but is not in any distress. Laryngeal examination with small Jackson laryngoscope shows a rather thick, stubby epiglottis and a redundancy of the mucous membrane of the right arytenoid. There is some question of infection of the right crico arytenoid cartilage, which is hard to rule out at present."

The second case is one of a small group of six patients who presented themselves at the clinic because of hoarseness. Several of these had complained of this symptom only since the removal of their tonsils, though in none of them was there any local evidence in the throat to explain the hoarseness. Laryngoscopy in these patients universally failed to reveal any gross pathology such as papilloma or polyp, the appearance being simply that of a congestion of the mucous membrane of the glottis, a failure of complete approximation of the cords and an increased coughing reflex. No ulcerations could be found and the possibility of tuberculosis seemed an unlikely one. One hesitates al-

ways to make a diagnosis of simple acute or chronic laryngitis until all other possibilities have been carefully excluded and it was for just this reason that laryngoscopy was deemed advisable. With a few of the older children tact and patience were rewarded by a good indirect view of the larynx but a direct one was always more satisfactory. With a careful and at the same time rapid examination it was often surprising to find children returning for subsequent laryngoscopies with actual willingness and little fear of the procedure.

CASE II. "Boy, J. H., age 6 years. Complaining of hoarseness for two months. Laryngeal examination shows both cords moving normally and symmetrically and of normal color and conformation. Both arytenoids are swollen, red and slightly edematous, as is also the intra-arytenoid space. Covering the mucosa is a small membranous patch. Culture negative for diphtheria. Question of tuberculosis or chronic laryngitis. Treatment consisted of enforced vocal rest and an alkaline spray. Subsequent laryngoscopies showed a definite improvement in the appearance of the larynx."

The third case represents a somewhat similar type of laryngeal pathology but of a more marked degree, since the element of respiratory obstruction is introduced. Such cases must be laryngoscoped to determine or exclude the presence of a foreign body in the larynx, whether there be any actual history of aspiration or not. Tracheotomy if needed should not be postponed, since early and tranquil operation is far easier and more satisfactory than one done at the last moment as a life saving procedure. In this instance the actual cause of the laryngeal edema was not satisfactorily determined, owing to the development of measles and necessity of sending patient to contagious hospital.

CASE III. "Boy, J. G., 3 years of age. On admission patient showed a good deal of inspiratory dyspnea with substernal retraction; not cyanotic. Nothing pathological appeared in the throat itself. Direct laryngoscopic examination, no anesthesia. The epiglottis and arytenoids were essentially normal. The cords seemed to be covered with a very thin exudate and to be much more closely approximated than normally. The whole laryngeal lumen narrowed down just like a funnel toward the trachea. It was impossible to introduce a bronchoscope of any size through the larynx. Tracheotomy considered and postponed. Somewhat more dyspneic in the morning and tracheotomy done with entire relief to dyspnea. Uneventful convalescence till development of measles."

Gradual development of a laryngeal stricture is represented by the fourth case. In this instance tracheotomy was followed by dilatation of the laryngeal lumen from above by means of triangular shaped sounds introduced at weekly intervals until a normal lumen for the child's age was obtained. The tracheotomy tube was then left in place for a considerable time and finally removed.

CASE IV. "Boy, M. F., 4 years and 7 months of age. Negative family and past history. Patient came into hospital complaining of having had difficulty in breathing for past two years. This has been evidenced by stridor and shortness of breath. Tonsils and adenoids removed without relief. For last two months difficulty in breathing has increased. No history of aspiration of foreign body.

"Laryngoscopy without anesthesia. Epiglottis normal. There is marked swelling and edema of all the supra-glottic structures with great narrowing of the glottic lumen. There is no impairment of cord motility. No reaction from operation. No change in voice. Second examination started under ether but increased respiratory embarrassment immediately followed, affording a strong argument against the use of an anesthetic in these examinations. Later tracheotomy done and a Number 2 tube inserted. After two weeks' stay patient was sent home from hospital. Has since been dilated by sounds up to 18, 24 and 26. The larynx has a more normal appearance and the voice, even with the tube in place, is stronger. Some slight difficulty in sleeping. Gradual corking of the tube was begun and after three weeks a full cork was easily tolerated. Two months later tracheotomy tube was removed and the wound allowed to close. Condition much improved, though voice still slightly husky."

Any considerable experience with diseases of the upper respiratory tract must occasionally lead to a tragedy. The fifth case proved to be one of a most distressing type in spite of every effort made to avert it. It exemplified in the strongest way that young children do not withstand any real degree of respiratory obstruction without becoming profoundly exhausted and tracheotomy under these circumstances should never be delayed. The development of subcutaneous emphysema following this procedure was a most unexpected sequel and one which post-mortem examination failed to explain satisfactorily.

CASE V. "Boy, W. S., age 18 months, admitted to hospital for prolonged coughing and beginning laryngeal obstruction manifested by gradually increasing substernal and supraclavicular retraction. Suggestive history of possible aspiration of piece of popcorn. Laryngoscopic examination with removal from the larynx of very small piece of white material like a macerated corn shuck. Hasty examination of the trachea and bronchi with 4 mm. bronchoscope, no anesthesia. No further evidence of foreign body and breathing through the bronchoscope entirely free. Shortly after examination respiratory obstruction increased so much as to necessitate tracheotomy, which was particularly difficult owing to the thickness of the pretrachial tissues. Shortly after this subcutaneous emphysema began to develop in the neck and gradually extended all over the body. Death ensued eight hours later.

"Post-mortem examination failed to reveal any gross break in the bronchial tree through which the air could have gained access to the subcutaneous tissues."

In practically all of these cases the larynx alone was the seat of trouble. In some of those classified below as bronchoscopic there was an initial suspicion that the trouble was in the larynx, but since in these the problem became ultimately a bronchoscopic one they are listed as such. Of course every bronchoscopy is initially a laryngoscopy.

The sixth case, that of an adult, is of interest in that the diagnosis lay outside of the usual

causes of hoarseness at this age. Tuberculosis and syphilis were excluded. The appearance did not suggest malignancy and the subsequent course of the disease strongly supports the opinion that it was a benign chronic inflammatory process.

CASE VI. "R. M., age 22 years, complaining of hoarseness without dyspnea for six months. A small, irregular mass was seen by indirect laryngoscopy protruding from the central portion of the left cord. This was removed six months ago, following which there was distinct improvement. Hoarseness recurred and direct laryngoscopy was done. Larynx exposed and cords found to be thick, irregular and roughened, poor approximation. An ill-defined mass was seen in the centre of the left cord. With laryngeal tissue forceps the irregular areas on both cords were removed in pieces too small for microscopic examination. A week later patient reported that voice was much better, though still a little rough. Cords appeared somewhat ragged by indirect laryngoscopy, but the mass on the left side was no longer present. Ten days later voice was still better."

BRONCHOSCOPIC GROUP

Of late years the field of bronchoscopy has been extended well beyond that of the removal of foreign bodies. More and more its use is indicated in the diagnosis of obscure pulmonary lesions and unexplainable cough and as a therapeutic adjunct in the treatment of chronic lung suppuration. The following series of cases show the lines along which this procedure has been carried out through the year.

Foreign bodies in the bronchi will always stand first in order as an indication for bronchoscopy. Their successful removal is spectacular, their demand for skillful technique extreme. Unfortunately removal of foreign bodies is not always curative, particularly when the condition has remained unrecognized and untreated over a long period of time. Such a case is the eighth, in which lung suppuration following the 15 months' sojourn of the foreign body in the bronchus was so extensive that bronchoscopy, although successful so far as foreign body removal was concerned, failed to cure the patient.

CASE VIII. "Girl, F. C., 3 years old. Family history and past history negative. Fifteen months ago child developed a fever, with coughing and expectoration. She was seen by a physician, who made no diagnosis for about a week, at the end of which time he said she had pneumonia. About the middle of the third week of her illness she was definitely better, but relapsed into a state of chronic coughing and intermittent fever. This condition has persisted until the present time. About three weeks ago she began raising large amounts of foul green pus, particularly in the prone position.

"X-ray shows a metallic wood screw in the right main stem bronchus as well as air and fluid in the pleural cavity. Diagnosis: Foreign body in bronchus, pyopneumothorax, lung abscess.

"Bronchoscopy, no anesthesia. Larynx exposed and 5 mm. aspirating bronchoscope inserted. Occluding the right main bronchus was a small, dark object, which after considerable aspirating of pus was seen to be the presenting head of the screw. Forceps were applied and the screw drawn into the bronchoscope and removed. Child in excellent condition.

"Three days later began expectorating a good deal of foul pus, rales appearing in the left side of the chest. General condition not quite so good. Thoracotomy done without avail and death ensued. No post-mortem could be obained, but suspicion remains that removal of the screw uncorked a lung filled with pus, which was then aspirated into the left lung, causing a pneumonia. Perhaps thoracotomy before bronchoscopy would have avoided this."

Non-opaque foreign bodies introduce an additional element of difficulty in diagnosis since X-ray does not afford the definite information so useful in cases of metallic foreign body.

The next case is one of two instances in which peanut kernels were removed, full reports of which were published in the BOSTON MEDICAL AND SURGICAL JOURNAL for July 30, 1925. A brief summary of one of these only is given.

CASE IX. "Boy, R. B., age 2 years. Patient admitted for removal of foreign body in the bronchus. First laryngoscopic examination showed considerable laryngeal edema. Bronchoscopy postponed for two weeks. March 31, 4 mm. bronchoscope introduced into the left primary bronchus, from which a small fragment of peanut was removed. This was followed by severe choking a few hours later, necessitating second bronchoscopy, at which time the remainder of the kernel was removed. Laryngeal edema developed subsequently, necessitating temporary intubation. One week later laryngeal edema recurred, requiring tracheotomy. Gradual decanulation, removal of the tracheotomy tube and closure of the tracheal fistula."

All symptoms and signs including the history may point to the presence of a foreign body in the respiratory tract and still this may be absent. The tenth case represents a very serious and often puzzling condition which can best be described as a laryngo-tracheo-bronchitis. The initial symptoms are very suggestive of foreign body, the more so when as in this case there is a history of possible aspiration. Other similar cases have been reported, several of which were likewise fatal. Apparently the accumulation of tenacious muco-pus in the lower bronchi acts as a complete obstruction, though all means possible to the extent of removal by bronchoscopy through the tracheotomy wound, as in a case reported by Lynch, have been used to combat it.

CASE X. "Baby, F. L., age 2 years. Was in perfect health until three days ago when she picked up a piece of nut shell from the floor and presumably swallowed or aspirated it. Since that time she has been in acute respiratory distress, but has eaten without difficulty. During the last 24 hours baby has become definitely weaker and breathing more and more labored.

"When first seen child presented a typical appearance of acute laryngeal obstruction with harsh stridor and substernal retraction. No cyanosis.

"Operation, laryngoscopy, no anesthesia. Larynx exposed. Some edema of arytenoids. In the midline below the cords appeared a white, flat structure about 1/16 inch across and the width of the lumen anterio-posteriorly. Efforts to grasp this with laryngeal forceps, however, brought out nothing, and after

a second attempt the child's condition became essentially normal, stridor ceased, also substernal retraction. Another view of the larynx disclosed absence of the object seen before. The possibility of its having been pushed down into the trachea was a strong one. Child put to bed and slept quietly all night, when, following a coughing spell, exactly the same symptoms of obstruction appeared as on admission.

"Tracheotomy, local anesthesia. Number 1 tube inserted, with relief of all symptoms of obstruction. Good condition except for temperature of 103. Some obstruction with the inner tube in place, relieved by removal.

"Laryngoscopy, no anesthesia. The larynx appears more swollen, no definite foreign body can be seen. The whole glottis is narrowed, small patches of exudate appear just below the cords, but the structure suggesting a foreign body initially is no longer present. All efforts to come in contact with any solid object even below the cords were unsuccessful. The 4 mm. bronchoscope could not by any possibility be introduced through the glottis, but an attempt at its insertion produced a few pieces of what looked like membrane. This was cultured for diphtheria. Child put back to bed after aspiration of the outer tracheotomy tube with Jackson's copper aspirator. Diphtheria antitoxin was administered.

"Some difficulty with tube clogging up during next night. When seen in morning temperature 105, respiration 60. Good color. Question of pneumonia. Seen again later and appeared to be in better general condition. Very little, if any, expectoration. In afternoon respiration became more rapid. Medical consultation reported signs of right bronchial obstruction. Condition became generally worse and child died next morning."

Body taken by Medical Examiner, who reported, verbally, no foreign body. Diphtheria cultures were negative. A streptococcus laryngotracheitis and bronchitis seems to have been the most probable diagnosis. Apparently the thick unexpectorated bronchial secretion collected to form obstructing plugs in the terminal, branch and main bronchi.

The following case is of the same nature, with, however, a favorable outcome and a less severe infection.

CASE XI. "Baby, M. D., age 18 months. Protracted story of intermittent coughing with recent stridor and change in voice. No cyanosis. All general medication failed to relieve the trouble. The first bronchoscopic examination revealed an edematous trachial mucous membrane with purulent secretion apparently coming from both main stem bronchi. Diagnosis rested between infectious tracheitis and bronchitis and external tracheal pressure from glands. X-ray showed no evidence of lung abscess, foreign body or enlarged thymus. Treatment consisted of bronchoscopic aspirations and instillation of gominal into the bronchial tree. Benzoin inhalations at home. After three treatments the secretion became greatly lessened and the wheezing character of the respirations disappeared. Parents report no further symptoms."

A group of eight patients treated for some form of chronic lung suppuration is represented by Case XII. It is only within the last few years that this long standing and distressing condition has been treated by bronchoscopic aspiration, removal or cauterization of obstructing granulations and instillation of some form of astringent and healing medication. Much has been written in favor and no little against this treatment. The chronicity of lung suppuration is such that immediate results rarely follow any form of treatment and only a large series of cases followed carefully over a long period of time can furnish trustworthy statistics. Needless to say, where repeated bronchoscopies are done on one patient, they must be performed as expeditiously, as painlessly, and still as thoroughly as possible. This is particularly true of adults. Children seem much more tolerant and without anesthesia will return week after week for treatment, showing no fear of a procedure which they have already experienced and requiring little if any restraining.

CASE XII. "Boy, K. G., 8 years old, comes in to hospital complaining of recurrent bronchitis every winter since infancy. Whooping cough three months ago, since when he has had a spasmodic cough at intervals, sometimes followed by attacks of vomiting. X-ray showed density at right base, suggesting an abscess of the lung. During stay in hospital patient coughed frequently, sputum being thick, yellow, and foul in odor.

"First bronchoscopy was unsuccessful because of vomiting. A second bronchoscopy showed purulent secretion coming largely from the right main bronchus.

"Following two more bronchoscopies in which purulent material was aspirated from the right lung, X-ray showed a distinctly clearer lung field at the right base with marked fibrous changes clearly defined in a way not seen on admission. A note a month later says general condition seems good. Very small amount of purulent material aspirated from right side. To go two or three weeks without bronchoscopy. X-ray shows right basal density much less marked.

"Returning after no treatment for two weeks patient showed a more profuse purulent discharge from the right lower lobe. Thorough aspiration, following which patient reported no symptoms and less cough. Began attending school and seemed to feel generally well. Next examination showed moderate amount of purulent secretion without odor. Thorough aspiration, injection of gominal. Bronchoscopies have been continued at two-week intervals and at the last one, a month ago, the bronchi appeared entirely free from purulent secretion."

In the few cases thus treated by the author the results have been varied. Case XII has definitely improved and gives promise of a cure. "A child of three with a post-tonsillectomy abscess of the lung is clinically much improved, though a certain amount of coughing and expectoration still persists. She has been bronchoscoped seven times with intervals of from a week to a month, during which general hygienic measures and postural drainage have been carried out, resulting in better sleeping, eating and all round health."

"An adult girl of 23 has been treated during the year for a post-tonsillectomy upper left lobe abscess, for which an unsuccessful rib-resection had been performed. After half a dozen bronchoscopies there was an extraordinary improvement, with almost complete cessation of purulent expectoration, gain in weight and ap-

petite and a return to light work. Bronchoscopies were stopped. A month later patient reported that she had taken cold, had had a severe pulmonary hemorrhage, and was losing weight. Repeated efforts to establish a diagnosis of tuberculosis have been unsuccessful, but patient has suffered a marked and extensive recurrence of her pulmonary infection and is doing poorly. No further bronchoscopic treatments have been carried out. More recently her general condition has improved and great hope is held for her ultimate recovery."

A case of long-standing bronchiectasis in a war veteran was treated a few times without any appreciable change in the condition. This patient, far from objecting to bronchoscopy, actually anticipated his treatments and was disappointed when they were discontinued.

Two other patients with pulmonary infections were bronchoscoped diagnostically to rule out foreign body or malignancy. Further treatment did not seem advisable, since on both spontaneous drainage seemed to be adequately taking care of the abscess. Bronchoscopy served to establish the diagnosis in two other cases of pulmonary malignancy manifested by peribronchial changes and encroachment on tne bronchial lumen.

Pulmonary infections still present therapeutic problems that are unsolved by the bronchoscope. It is certainly a diagnostic aid in all cases and frequently serves to substantiate what can only be suspected on physical examination or X-ray. It is a definite therapeutic agent in a certain number of cases, and it is plain that the average patient would not submit to repeated treatments unless they felt themselves benefited by them. In the author's limited experience there are an equal number of cases which do not respond noticeably to bronchoscopic treatment. On the other hand, no other form of treatment can cure all these patients, many of them are not suitable for surgical intervention, and general medical treatment likewise seems unavailing. Under such circumstances one turns to bronchoscopy as a procedure which may possibly be of some slight help in these distressing conditions. Roentgen-ray examination following the bronchoscopic introduction of opaque mixtures such as Lipiodol is a further step toward shedding some light on this problem.

Further use of the bronchoscope was made in an effort to obtain pure bronchial cultures in three cases of bronchial asthma, in the hope that an uncontaminated vaccine might thus be secured. This work is being carried on in connection with the pediatric department and is still unfinished. Finally bronchoscopy was employed in a few instances of unexplained or prolonged cough. Such an example is the thirteenth case, in which evidence of pretracheal pressure was definitely observed. A similar case of mediastinal tuberculosis, age eight weeks, was bronchoscoped and diagnosis later confirmed by post-mortem examination.

CASE XIII. "Girl, J. M., 2 years and 9 months, admitted to the hospital with a history of acute illness of 10 days' duration and an intimate exposure to tuberculosis. She continued to run an irregular temperature during her stay. Her intradermal tuberculin was strongly positive. There were coarse consonating rales audible, scattered, through both chests during her stay.

"Bronchoscopy, without anesthesia. Five millimeter aspirating bronchoscope introduced into the trachea. The trachial walls were edematous and swollen irregularly, producing folds in the tracheal lumen, which is greatly narrowed and deformed. Further down the main stem bronchi the walls were not round in shape and appeared constricted and swollen. No reaction."

Thus it would appear that even in a small group of cases the bronchoscope has a definite place as a diagnostic and therapeutic agent and with careful and discreet usage is not a dangerous or drastic instrument.

### ESOPHAGOSCOPIC GROUP

Technically esophagoscopy presents as much of a problem as bronchoscopy in spite of the fact that only a single instrument is used. The position of the patient's head is extremely important since on it depends the overcoming of the muscular spasm at the upper end of the esophagus, where the crico-pharyngeus muscle offers at first considerable impediment to the introduction of an instrument into the thoracic esophagus. Folds of mucous membrane and the presence of various fluids and secretions make a good inspection of the esophagus less satisfactory than of a bronchus whose rigid walls and regular lumen are much more readily seen. Hence foreign bodies are more easily overlooked and application of forceps rendered more difficult.

Seven foreign body cases were included in the year's work. A brief account of each will be given.

CASE XIV. "Boy, A. M., 2 years and 7 months, came to the hospital with history of being seen playing with a five-cent piece, after which he began to vomit repeatedly. Inability to find the coin aroused parents' suspicion that it had been swallowed. X-ray taken, showing coin in upper esophagus.

"Esophagoscopy, ether anesthesia. Child's size esophageal speculum inserted behind cricoid and the upper end of the esophagus exposed. Coin seen lying transversely, grasped with laryngeal grasping forceps and removed. No trauma. Discharged home two days later."

CASE XV. "Boy, J. M., with foreign body shown by X-ray in upper esophagus at suprasternal notch.

"Esophagoscopy, no anesthesia. Child's size esophageal speculum inserted, larynx displaced forward, and upper end of esophagus exposed. A penny seen plainly, lying transversely. Edge easily grasped with laryngeal grasping forceps. Removal and uneventful convalescence."

CASE XVI. "Boy, H. W., age 1½ years, came to hospital with history of having swallowed a penny three days ago. Since then has been fussy and has vomited almost everything that he has taken. X-ray shows a penny in esophagus.

"Esophagoscopy, without anesthesia. A child's esophageal speculum was inserted in upper esophagus and the edge of the coin exposed. It was covered with orange pulp and had a peculiar light gray appearance. The edge was grasped with laryngeal grasping forceps and the foreign body removed. Child discharged home in good condition."

These three cases represent a very frequent type of foreign body in children; namely, the coin. Most of these lodge at the upper end of the esophagus and appear on X-ray at or near the sterno-clavicular junction. They can usually be reached by use of an esophageal speculum, and, having no sharp points, be removed without much difficulty.

The following case illustrates the necessity of an X-ray or fluoroscopic examination immediately before attempted removal. Here three days elapsed between the X-ray and operation, during which time the coin undoubtedly slipped into the stomach. Since this case we have had all such foreign body cases fluoroscoped immediately before operation.

CASE XVII. "Girl, I. M., age 4 years, admitted to hospital with history of having swallowed a nickel. No symptoms except that she says throat hurts on swallowing. X-ray shows coin about midway between throat and stomach.

"Esophagoscopy, no anesthesia. The child's esophageal speculum was passed as low as possible behind the larynx, but no foreign body was seen. The 7 millimeter full lumen esophagoscope was then introduced and the esophagus carefully exposed as far as the cardia without any sign whatsoever of the coin. X-ray one hour after operation shows the coin in the duodenum.

"No symptoms; eating solid foods without trouble. To have stools watched for coin and to be X-rayed again."

In this instance the coin was below its usual point of lodgment and hence was far more likely to slip into the stomach than if held by the spastic esophageal muscle higher up.

One of the most difficult problems with which the esophagoscopist has to deal is that of the open safety pin, particularly when the latter is present point upward. Great care must be exercised to prevent the point perforating the esophageal wall and causing a mediastinitis. Among the various methods and instruments used in this work, all aim to protect the bared point of the pin. Some instruments, such as the ingenious one devised by Mosher, close the pin entirely, others allow the blunt keeper to ride outside the esophagoscope. The actual technique of removal is beyond the scope of this paper, but the three safety pin cases of the year will be reviewed.

CASE XVIII. "Patient, 9 months old, brought to hospital two hours after accidental swallowing of open safety pin. X-ray shows pin open in the upper esophagus, lying transversely with the point to the left.

"Esophagoscopy, no anesthesia. Upper esophagus exposed by drawing the larynx forward with the laryngoscope, thus bringing into view the keeper on the right and shaft of the pin on the left. Spring was not seen and the point was slightly buried in the pharyngo-esophageal wall. The lip of the esophagoscope was rotated to the left in such a way as to form a fulcrum near the point of entrance of the pin into the mucous membrane. The keeper was then grasped with forceps and pulled through the lumen of the laryngoscope, thus straightening out the pin, while the point was prevented from further ingress by the rigidly held lip of the laryngoscope. Convalescence was uneventful."

CASE XIX. "Two-months-old baby, in the hospital for treatment of eczema, suddenly had a profuse hemorrhage from the mouth. Immediate X-ray revealed an open safety pin, point downward in the upper end of the esophagus. Immediate esophagoscopy was performed without anesthesia, though the child at this time was in an extremely critical condition from loss of blood. The pin, with its point firmly imbedded in the esophageal wall, was quickly extracted by grasping the spring and withdrawing the whole pin with the point trailing. No further bleeding. Immediate transfusion was performed, in spite of which death ensued on the following day."

CASE XX. "Child, 4 years old, presented himself at the hospital with an open safety pin lodged point upward just above the diaphragmatic opening. A slotted 6 millimeter esophagoscope was inserted, the pointed shaft of the pin located and grasped with pin forceps. The point was imbedded in the esophageal wall to such an extent that the downward pressure necessary to free it forced the pin into the stomach. It was later removed by gastrotomy and an uneventful convalescence ensued."

### ESOPHAGEAL STRICTURE

No more tragic accident can happen to the digestive tract than the accidental swallowing of caustic or lye. In spite of all propaganda some parents still regard these cleansing materials as harmless, and leave them in places where children can secure and swallow them. The result is frequently a marked cicatricial stenosis of the esophagus which requires months and years of painstaking and patient treatment before anything like normal conditions can be restored.

The methods of treatment of lye stricture are numerous and have been dealt with by various authors. In many of them esophagoscopy plays a most important part, since by its use direct vision of the stricture is obtained and dilating bougies can be introduced with the minimum risk of trauma. The latest and apparently safest method consists in the use of the Tucker retrograde bougies. In four cases included in this series, two were treated by this method and are still under treatment. Progress has been satisfactory and the results so far obtained give promise of an eventual cure. One case was found to have so little trauma of the esophageal wall that no treatment was needed. The fourth case, in which dilatation was first carried out by the use of olive-tipped bougies, finally came to a gastrostomy. Some weeks later, when a string was being introduced preparatory to undertaking retrograde bouginage, the patient unfortunately developed a peritonitis and died, termination in no way attributable to this form of dilatation.

### "CARDIOSPASM"

Brief mention should be made of two cases of so called cardiospasm, both occurring in young adults who for years had been totally unable to enjoy normal eating and digestion. X-ray examination in both showed a typical low diaphragmatic constriction of the esophagus with enormous dilatation of the esophageal wall above. In the first patient treatment was carried out by esophagoscopy and the introduction under direct vision of bougies up to a size incapable of passing the esophagoscope. As a result a distinct improvement was noticed by the patient, she regurgitated less, was able to enjoy eating more than at any time previously and began to gain weight. With dilatation carried thus up to a No. 24 bougie in the absence of any pouches or diverticula it was felt safe to continue dilatation by the passage of bougies by mouth. This was carried out up to No. 34, with increased improvement in every way, when suddenly, following the passage of a No. 36 bougie, definite signs of acute abdominal pathology appeared and a laparotomy was performed. Nothing was found but a hematoma along the lesser curvature of the stomach, presumably due to the rupture of a vein near the cardia. A slow but uneventful convalescence ensued, but further dilatation has been discontinued. This complication serves to emphasize the teaching of Trousseau that bouginage must in any circumstances be undertaken with the utmost caution.

In the second patient, diagnostic esophagoscopy, which demonstrated a marked degree of stasis and esophagitis, was followed almost immediately by gastrostomy, for the purpose of putting the esophagus at rest and affording the patient the maximum amount of nourishment. Later a string was swallowed and retrograde bouginage with Tucker bougies was carried out up to No. 30. The result so far has been a marked improvement in the general condition with comfortable eating and a gain of twenty pounds in weight.

A few other diagnostic laryngoscopies, bronchoscopies and esophagoscopies have made up the total of the year's work. Some of the results have been disappointing, most of them have been satisfactory. The records are presented here in the hope that the general picture may be helpful to other workers in this field who, handicapped by the rarity of these cases, are nevertheless called upon from time to time to deal with them.

## THE VALUE OF SPECIFIC TREATMENT IN CARDIOVASCULAR SYPHILIS*

BY BERNARD I. GOLDBERG, M.D.

THERE is a belief prevalent among some clinicians that the specific treatment of cardiovascular syphilis, when present to the degree that symptoms or demonstrable signs exist, is of little avail. The doubts expressed are based upon such factors as failure of results in certain cases, particularly advanced cases and aneurysms, and apprehension regarding its safety, for dangerous and rapidly fatal issues have been reported. This belief is furthered by the results of misapplication of antiluetic treatment. It is my purpose in this paper to counteract this attitude.

### PROPHYLAXIS

Obviously cardiovascular syphilis can be greatly averted by the prompt treatment of the disease during the primary and secondary stages.† This connotes intensive treatment, for Warthin[1] has clearly demonstrated in his pathological studies how frequently a cure is but an illusory one and the patient is in a latent syphilitic state, proven by the presence of spirochetes in the myocardium and aorta.

It is now well established that with the initial infection the spirochetes become disseminated throughout the tissues, including heart and aorta. As a result of painstaking, microscopical researches dating from 1906[2, 3, 4, 5, 6], the spirochetes have been found in the tissues in relatively few months after the initial infection as well as many years later. Not infrequently serious cardiovascular disturbances occur long before the proverbial so-called tertiary stage. Brooks[7] has reported a case of perforation of the aorta before the secondary stage had been fully developed. Keeping these contingencies in mind, one will not be misled by th statistics that the average duration of time is ten to twenty years before the onset of symptoms. Reid[8] has found at the Massachusetts General Hospital that an average of sixteen years elapsed before the onset of definite symptoms.

The frequency of syphilis combined with the more or less spirochetal predilection for the cardiovascular system accounts for the large percentage of cases seen in clinics and at the autopsy table. In any large autopsy service cardiovascular syphilis occupies a prominent position. Oberndorfer[9] reports 7 percent of 1437 autopsies and Gruber[10] found 4½ percent in 6000 autopsies. Reid[11] noted 54 cases in 1678 autopsies or 3½ percent at the Massachusetts General Hospital. The literature[12, 13, 14, 15] is replete with statistics from various clinics emphasizing the role syphilis plays in cardiac

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†The sharp demarcation between the stages in syphilis is now regarded as more apparent than real.

fatalities. Certainly a large number of these cases reflect directly the inefficacy of earlier methods of treatment and syphilographers have been quick to respond to the lesson gained.

I shall traverse the question of diagnosis by merely stating that with the aid of a careful history, a complete physical and cardiovascular examination and X-ray a diagnosis even in fairly early cases may often be established.

### THE WASSERMANN REACTION

The outcome of the Wassermann reaction has been unduly stressed in determining the presence or absence of cardiovascular lues. The Wassermann may be negative in the early and most sought for cases where doubt hovers over the diagnosis yet these are the cases most amenable to treatment. There is also a fairly large percentage of cases where in spite of clear cut evidence of the presence of aortitis, aortic regurgitation or aneurysm the Wassermann is repeatedly negative. Warthin[16] has actually demonstrated spirochetes in many of the Wassermann-negative cases. The reader is referred to the literature[17], [18], [19], [20], [21], [22] for statistics emanating from the various clinics regarding the percentage of positive Wassermanns in proven cases of cardiovascular syphilis. In our clinic we have come to pay less attention to the Wassermann and more to the other factors in diagnosis.

### TREATMENT

The treatment of syphilis is a prolonged affair irrespective of stage or type of lesion. Roughly it may be stated that the sooner treatment is instituted following initial infection, the more intensive can be the treatment at the outset and the safer this form of treatment. Persistency in treatment however cannot be stressed too strongly if one wishes to secure results. In the attack on cardiovascular syphilis we have at our disposal the same armamentarium as is available in the treatment of the other manifestations of the disease. This includes the arsenicals, mercury, bismuth and potassium iodide. The great moot question has revolved about the method of administration.

### ARSENICALS

The arsenicals are apparently the dangerous though our most effective weapons. Their use is of paramount value in early cases. The fear of the arsenicals has been partly justified by the catastrophic results occurring in occasional cases. These accidents however can largely be averted by careful selection of cases. Cases exhibiting marked circulatory failure, large aneurysms or in elderly people, should with exceptions be placed upon treatment with the other drugs. Wile[23], although impressed with the beneficial effects of intensive treatment in the great majority of cases, has witnessed two caess of ruptured aneurysms and acute dilatation and failure following intravenous arsenic. The exceptions however have been sufficiently glaring to command attention and warrant a trial of these therapeutic agencies.

### MERCURY, BISMUTH AND IODINES

Mercury in its various forms has been admittedly proven of value in all forms of visceral syphilis. It is given preferably intramuscularly into the gluteal muscles according to the technic described by Stokes[24]. One may use the salicylate of mercury or other preparations. Some prefer the inunctions which have recently been demonstrated to be an effective pathway of absorption[25]. More uncertain are the preparations taken by mouth as the protiodide or mercury with chalk. During the period of mercury medication one must ever remember the mouth hygiene and urine examinations.

Bismuth is one of our more recent therapeutic introductions. No detailed reports as yet have appeared relating to its specific effects on cardiovascular syphilis. Experimentally there is definite evidence that it has active spirochetocidal powers[26], [27]. Grund[28] who has recently reviewed the subject and Hopkins[29] consider it superior to mercury. That it is not a new therapeutic phantom is shown by the careful studies made and excellent results obtained in specific infections. It is not to be expected nor is it intended that it should replace arsphenamine. In cardiovascular lues as an adjunct to the other drugs it shows particular promise. Where one dislikes to use arsphenamine either because of intolerance or in contraindicated cases for other reasons bismuth may be used with a minimum of apprehension. Whether it will supplant mercury as the drug to be utilized between arsphenamine courses further studies will disclose. Its utilization in the so-called Wassermann fast cases is not particularly applicable to the type of cases which concern us here. The iodide of potassium still manages to retain its empirical value. We are in the same position now as they were a generation ago as regards any accurate knowledge of its action.

### TECHNIC OF ADMINISTRATION

Clinicians and syphilographers have repeatedly warned against standardization of treatment and advocated individualization. This dictum is obviously true. Yet in large clinics a nucleus of a standardized program is essential; the variations can be appropriately manipulated. The cases may be arbitrarily divided into early and advanced cases.

### TREATMENT IN EARLY CASES

Arsphenamine or allied arsenicals is the treatment par excellence in early cases. Rarely are cases of cardiovascular syphilis suscepti-

ble to diagnosis early enough for one to be secure in approaching the treatment of these cases as one does the chancre or secondary manifestations. Hence as a rule it is a better plan to begin with a short course of six mercury or bismuth injections given intramuscularly. We then proceed with weekly injections of intravenous arsphenamine* in initial doses of 0.15 gm. The tolerance to arsphenamine may be judged by the absence of either a Herxheimer reaction or an augmentation of cardiac symptoms and disability. If well tolerated the dosage may be increased until 0.4 gm. are given. About 6 to 10 injections should constitute a course. In the event of intolerance we are forced to resort to other forms of medication or decrease the dosage if the initial dose has been too large. Occasionally neo-arsphenamine is tolerated where arsphenamine is not and is often worth a trial. Deviations from this routine are of course a matter of judgment. Thus one may in some cases introduce arsphenamine at the outset where one feels secure that the balance is positive enough to warrant it. Or on the other hand one may give a larger initial dosage. Concomitantly the patient is given potassium iodide in increasing doses reaching one hundred grains or more daily. Then follows a rest period of six weeks to two months. Two, three, four or more courses are given according to indications.

### TREATMENT IN ADVANCED CASES

Those cases having reached a moderately advanced or advanced state require more caution. Courage, however, should not wane for one is often surprised at the happy results ensuing. As previously mentioned the use of the arsenicals is not so wide-spread in these cases, yet is not to be wholly ignored. To compensate the courses of mercury or bismuth are prolonged. It is in these cases where symptomatic relief is so often a godsend.

One feature too often neglected is the therapeutic test. This consists of four to six injections of arsphenamine and 8-10 intramuscular injections of a mercury preparation. Certain syndromes such as angina pectoris, arrythmias of various types, aortic dilatation, etc., often have a specific basis. The blood serology being negative many of these cases are given only symptomatic or direct cardiac therapy. Spectacular results have occasionally followed antiluetic treatment.

It is beyond the scope of this paper to discuss the direct cardiac therapy, such as the use of digitalis, etc.

### EFFECTS OF TREATMENT

Probably the most disconcerting feature of this condition is the agonizing pain often experienced by these patients. The pain may be typical or atypical, as Willius[30] has graphically shown. The similarity of the manifestations of angina pectoris, whether of specific or nonspecific origin, should draw our attention to etiologic therapy. As has been mentioned the Wassermann is likely to be negative. In the cases of specific origin, there is often a remarkable amelioration of symptoms following treatment, the pain or pressure sensation being either diminished or entirely abolished. As few as two or three injections of arsphenamine have occasioned relief from pain in some cases.

Heart block unexplained on a rheumatic or arteriosclerotic basis is usually a syphilitic sequela and as such should receive the benefit of at least the therapeutic test. The same may be said of heart failure in a patient, especially an adult in the third and fourth decades, without a rheumatic history. Unusual degrees of benefit are often derived in these cases as a result of treatment. Vaquez[31] states that the elimination of attacks of syncope constituting the Stokes-Adams syndrome has been almost entirely limited to treated luetic cases. In the cases of cardiac insufficiency uncomplicated by heart block or aortic regurgitation or long standing broken compensation a therapeutic test, limited though it is, has often yielded surprising results. Brooks[32] reported that two of five cases of heart block with active treatment showed relief from "symptoms of dyspnea, cardiac pain and defective capillary circulation." Longcope[33] concludes in an excellent study that the precordial pain, paroxysmal dyspnea and angina pectoris are temporarily or permanently relieved by repeated injections of arsphenamine. Anderton[34] and Koppang[35] feel that symptomatically the results have been gratifying. Howard[36] states that many cardiac abnormalities have disappeared under treatment and even the most serious later involvements frequently respond very satisfactorily to specific treatment. Energetic treatment is advocoted by Cumston[37] who is impressed with the results. Eliot[38] is very emphatic in his advocacy of specific treatment. In an analysis of 300 cases, Brooks and Carrol[39] noted definite improvement in a large percentage of cases following specific treatment.

The systemic effect of treatment must not be lost sight of. Cardiovascular lues in any case may dominate the picture but quiescent involvements in other organs are undoubtedly equally benefited. Thus endarteritis of the peripheral vessels, especially cerebral, may be prevented from progressing to actual thrombosis or hemorrhage. The bodily improvement is also often noticeable.

The duration of life is not regarded as considerably prolonged except in very early cases. Reid[40] found at the Massachusetts General Hospital that after the establishment of the diagnosis of aortitis the length of life is about three to one in favor of those cases receiving arsphen-

*Similar arsenicals are included wherever the word arsphenamine is used.

amine. This coincides with the general findings in other clinics.

The mechanism of the restorative process is apparently a replacement of diseased tissue by fibrosed and scar tissue. Considering the cardiac reserve as a quantitative function of the amount of normal muscular tissue, then the checking of the pathological process perhaps leaves sufficient well functioning tissues to allow ordinary activity.

### UNTOWARD EFFECTS

The untoward effects are almost wholly confined to the arsenicals. The nitritoid reactions occur less frequently in these cases because of smaller dosage. Stokes[41] states that arsphenamine "administers a therapeutic shock because of the rapidity of the healing changes it produces, and, because of its vasculotoxicity, is dangerous to bad hearts." Electrocardiographically[42] there is a slight prolongation of the conduction period and a shortening of the refractory phase. The fatal combination of ventricular tachycardia and fibrillation may be precipitated theoretically by the toxic action of arsphenamine, and perhaps this is what actually happens in those cases where collapse occurs following intravenous arsenic. It may not be amiss to mention here that relief from pain in angina has been sought by cervical sympathectomy. Admitting even grave risk in certain cases of cardiovascular lues, intravenous arsenic is hardly comparable to such a major operation.

### CONCLUSIONS

1. More efficient examination has disclosed a greater percentage of cardiovascular syphilis than was formerly believed.

2. The Wassermann is negative in an appreciable percentage of cases and does not rule out the presence of cardiovascular syphilis.

3. These cases should be subjected to a definite and persistent course of combined antiluetic treatment—such a course is here outlined.

4. Wherever possible arsphenamine or allied arsenicals should be given as it is far more effective than any other form of therapy.

5. Antiluetic treatment has been conclusively proven to have resulted in the amelioration of symptoms, especially the harassing pain of angina pectoris of luetic origin, and has been known to be of benefit in all cardiac deficiencies which have a specific basis.

### BIBLIOGRAPHY

1 A. S. Warthin: The Persistence of Active Lesions and Spirochetes in the Tissues of Clinically Inactive or "Cured" Syphilis. Am. Jour. Med. Sc., 152-508-1916.
2 Buschke and Fisher: Deutch. Med. Woch., 19-1906.
3 Simmons: Munch. Med. Woch., 5-1500-1906.
4 Rueta: Munch. Med. Woch., 53-778-1906.
5 C. Benda: Berlin Klin. Woch., 43-989-1906.
6 Schmorl: Munch. Med. Woch., 54-188-1907.
7 H. Brooks: The Heart in Syphilis. Am. Jour. Med. Sc., 146-513-1913.
8 W. D. Reid: The Heart in Modern Practice. J. B. Lippincott, 1923.
9 E. Oberndoffer: Munch. Arzt. Verein, Nov. 20, 1912.
10 C. Albutt: Diseases of the Arteries Including Angina Pectoris.
11 W. D. Reid: Specific Aortitis. Boston Med. and Surg. Jour., 183-67 and 105.
12 A. R. Lamb: Nelson L. L. Med., IV-533.
13 See Ref. 7.
14 T. Howard: Syphilis of Heart and Blood Vessels. Am. Jour. Med. Sc., 167-266-1924.
15 W. P. Anderton: Symptoms and Treatment of Central Vascular Syphilis. Boston Med. and Surg. Jour., 188-377-1923.
16 See Ref. 1.
17 W. T. Longcope: Syphilitic Aortitis. Arch. Int. Med., 11-15-1913.
18 C. G. Cumston: Aortic Pain and Antisyphilitic Treatment. Am. Jour. Syph., 8-349-1924.
19 U. J. Wile: Treatment of Syphilitic Liver and Heart. Am. Jour. Med. Sc., 164-415-1922.
20 Ref. 1.
21 Cummer and Dexter: The Relation of Aortitis to Syphilis and the Importance of Its Recognition. Jour. Am. Med. Assn., 59-412-1912.
22 W. C. Moore: Cardiac Syphilis. Am. Jour. Med. Sc., 155-660-1918.
23 Ref. 19.
24 J. H. Stokes: Technic of Intramuscular Injections. Med. Record N. Y., 99-18-1921.
25 H. N. Cole: J. G. Hutton and Torald Sollman. Jour. Am. Med. Assn., 82-199-1924.
26 A. E. Robert and B. Santon: Action du Bismuth sur la Spirillose des Poules. Ann. de l'Instit. Pasteur, 32-61-1916.
27 R. Sazeras and C. Levaditi: Etude de l'Action Therapeutique du Bismuth sur la Syphilis. Ann. de l'Instit. Pasteur, 36-1-1922.
28 J. L. Grund: Treatment of Syphilis with Bismuth. Boston Med. and Surg. Jour., 192-340-1925.
29 J. G. Hopkins: Antisyphilitic Action of Bismuth. Jour. Am. Med. Assn., 83-2087-1924.
30 F. W. Willius: Atypical Pain with Angina Pectoris. Med. Clin. N. Am., 371 (1921-1922).
31 H. Vaquez: Diseases of the Heart. W. B. Saunders, 1925.
32 Ref. 7.
33 Ref. 17.
34 Ref. 15.
35 N. B. Koppang: Norsé Mag. f. Laegevid., 83-65-1922 (abstr. by P. D. White, Boston Med. and Surg. Jour., 188-17-1923).
36 Ref. 14.
37 Ref. 18.
38 A. R. Elliott: Syphilis of the Aorta. Med. Cl. N. Am., 1-1305-1918.
39 Brooks and Carrol: The Treatment of Heart Involvement in Syphilis. Jour. Am. Med. Assn., 1914-63-1456.
40 W. D. Reid: Prognosis of Specific Aortitis. Jour. Am. Med. Assn., 73-1832-1919.
41 J. H. Stokes: Arsphenamine Treatment of Syphilis. Med. Cl. N. Am., 5-492-1922.
42 W. D. Reid: Toxic Effect of Arsphenamine on the Heart. Jour. Am. Med. Assn., 84-883-1925.

## THE OPTIC CANAL IN OPTIC ATROPHY

BY LEON E. WHITE, M.D.

As this paper is the third devoted to the investigation of optic canals, a brief resume of the preceding ones seems in order.

### ANATOMICAL

In an examination of about two hundred skulls the diameter of the optic canals was found to vary from 4 to 6 mm., the average being 5.17 mm., and the shape from the usually circular to various degrees of ovalness. Both size and shape depend upon the pneumatization within the accessory sinuses adjacent to the canals, the greater the pneumatization the smaller and more irregular they become. It seems probable that the neural contents of these small excessively pneumatized canals would be especially vulnerable, not only because they are more completely surrounded by the mucosa of

the adjacent sinuses but also because their bony walls are thinner and offer a lesser barrier to infections. Clinical data have fairly well borne out this belief.

### RADIOGRAPHS

The true size and shape of the optic canal can only be obtained when its axis is close to the lower outer quadrant of the orbit. The optic canal in the living can be thrown into this area when the patient is lying face down with the malar bone, nose and lower jaw touching the plate, the central ray directed straight down.

The diameter of the optic canal as it appears on the radiographic plate is enlarged about 10% but as this is the only method of determining its size no deductions are made in recording cases. The relative sizes are all that are needed. The normal canal in the radiograph is practically 5.5 mm. The average diameter, as obtained by filming twenty-five supposedly normal individuals, was 5.35 mm., and of these 10% were oval. These canals were contrasted with an equal number from patients with optic nerve involvement where the average diameter was found to be 2/3 mm. less and in 50% of these the canals were oval.

The conclusions from this anatomical and radiographic study are that if future cases substantiate the results of those analyzed, one can have a valuable guide as to the necessity for surgical intervention in cases of retrobulbar and optic neuritis.

### TREATMENT

In a second paper an attempt was made to ascertain what influence the size and shape of the canal might have on treatment. This was done by analyzing thirty-six cases previously treated for some form of optic nerve involvement. It was found that in the small canals, i. e., 4 mm. or less, there was 50% in which the loss of vision was permanent, while in the large canals this percentage was considerably smaller.

During the past two years an effort has been made to either substantiate or disprove these views. It seemed to me that by measuring the canals in a large series of optic atrophy cases some valuable data could be acquired. Accordingly through the hearty coöperation of the Social Service, the Ophthalmic staff, the X-ray Department of the Massachusetts Eye and Ear Infirmary, and the State Department of the Blind, many patients with optic atrophy were investigated. I personally made the nose and throat examination. The fundi were examined by a member of the Ophthalmic staff, usually Doctor Ellis, and the optic canals, sella turcica and accessory sinuses were filmed by Doctor A. S. Macmillan. Careful records were made by the Social Service, and at the completion of the work I analyzed the records made by the various examiners and have incorporated them in this paper. Many of the cases while diagnosed as optic atrophy had other eye conditions, but nearly all were helpful for comparison. While the main object of this investiation was to establish the relationship, if any, between the size of the canal and the loss of vision, another aim was to determine whether the canals became enlarged from increased intracranial pressure or narrowed when the nerve atrophied.

Sixty-seven cases of optic atrophy, mostly from the Perkins Institution for the Blind, were studied. Of these ten were unilateral. This gives 124 optic canals which are subdivided into five groups according to area. Occasionally the canals in a case differ so much in size that they belong in different groups. Either from the history of these cases or from previous records it has usually been possible to determine the cause of the atrophy. Whenever we have a specific history, or find Hutchinsonian teeth the atrophy is classified as of luetic origin, in spite of the fact that in many of these, infected sinuses, tonsils or teeth might have been a contributing factor. Many have had brain tumors removed or the radiographs show an enlarged sella. Here the atrophy is, in all probability, due to the intracranial condition and the nose and throat pathology is but an incident.

Cases which have been blind since birth or early infancy where the causes are not generally known, are classified as congenital. In these the nose and throat could not have been in any way a causative factor. In other cases the atrophy is the terminal result of a retinitis pigmentosa and the nasal condition has no bearing on the etiology. In some of the cases, however, the history is so typical of a retrobulbar or optic neuritis either from nasal, dental or tonsilar infection that there is little hesitation in classifying the atrophy as secondary to the neuritis. Several of these cases have, as a matter of fact, been followed from the acute onset to complete atrophy. 93 or 75% of the 124 canals associated with optic atrophy were below normal. 22 or 18% were normal. 11 or 7% were above normal.

### GROUP I

This contains the very small canals rarely, if ever, seen with normal vision. Their area is from 1/3 to ½ the normal. In this group is classed the 3, 3.5, 3.5x4, 3.5x5 and 4 mm. canals. thirty in all.

*Summary of these 30 canals:*

In 9 the atrophy was secondary to a retrobulbar neuritis of nasal origin.
All had obstruction to the posterior sinuses.

In 5 the atrophy was secondary to lues.
All had nasal blocking and suspicious tonsils.

In 6 the atrophy was secondary to brain tumors.
4 had nasal blocking or infected sinuses.
2 had infected tonsils.

In 6 the atrophy was congenital.
Nose negative in all.
Hypertrophied tonsils in 2.
In 2 the atrophy was secondary to retinitis pigmentosa.
Nose and throat normal.
In 2 the atrophy was secondary to spinal meningitis.
Hypertrophied tonsils.

*Comment:*

In eighteen of these thirty canals there was marked nasal pathology which in nine was considered to be the cause of the atrophy. While hypertrophied tonsils were found in eleven, in none were they considered to be a factor in the loss of vision.

GROUP II

These canals are somewhat larger than those in the preceding group and are found in cases with normal eyesight. Their small size, however, is a distinct menace to vision when their neural contents are involved in any inflammatory process. They are from 2/3 to ¾ of the normal size and comprise the 4x4.5, 4x5, 4.5, 4.5x5 mm. canals, forty-one in all.

*Summary of these 41 canals:—*

In 3 the atrophy was secondary to retrobulbar neuritis from nasal infection.
Infected sinuses.
In 6 the atrophy was secondary to chronic retrobulbar neuritis from dental infection.
Nose negative in all. Suspicious tonsils in three.
In 9 the atrophy was secondary to lues.
Nasal obstruction in 7. Tonsils suspicious in 5.
In 4 the atrophy was congenital.
In 2 pansinusitis, nasal obstruction and diseased tonsils.
In 4 the atrophy was secondary to retinitis pigmentosa.
In 2 nasal blocking and infected tonsil stumps.
In 2 the atrophy was secondary to infantile paralysis.
Nasal obstruction, pansinusitis, infected teeth and tonsils.
In 4 the atrophy was secondary to fractured skulls.
Nasal obstruction in all.
In 7 the atrophy was secondary to brain tumors.
Nasal obstruction in 5. Diseased tonsils in 3.
In 2 the atrophy was secondary to meningitis.
Nose and throat negative.

*Comment:*

In but three of these canals was nasal infection the probable cause of the atrophy, while infected teeth appeared to be responsible for the atrophy in six canals. The nasal obstruction and tonsilar infection found in most of the other cases were probably not responsible for the atrophy.

GROUP III

These canals are from ¾ to 9/10 of the normal size and include the 4.5x5.5, 4x6.5, 5 and 5x5.5 mm. canals, twenty-two in all.

*Summary of these 22 canals:*

In 2 the atrophy was secondary to multiple sclerosis.
Suspicious tonsils.
In 4 the atrophy was secondary to chronic retrobulbar neuritis of dental origin.
Diseased tonsils.
In 2 the atrophy was secondary to chronic retrobulbar neuritis from diseased tonsils.
Infected tonsils.
In 6 the atrophy was congenital.
Nasal obstruction and diseased tonsils in 2.
In 2 the atrophy was secondary to brain tumor.
Nasal obstruction, enlarged tonsils.
In 2 the atrophy was secondary to infantile paralysis.
Nose and throat negative.
In 2 the atrophy was unclassified.
Nose and throat negative.
In 2 unilateral cases the atrophy was secondary to acute retrobulbar neuritis probably from infected tonsils.
Nasal obstruction.

*Comment:*

In this group there are but two canals (unilateral cases) where the atrophy was thought to be secondary to an acute retrobulbar neuritis, probably of tonsilar origin, although in both there was some nasal obstruction.

There were six canals (three bilateral cases) where the atrophy appeared to be secondary to chronic retrobulbar neuritis of dental origin, although two of these also had suspicious tonsils.

In the other cases the nasal obstruction and suspicious tonsils did not seem to be a factor in the atrophy.

GROUP IV

These canals, twenty in number, are of normal size, the measurements being 5.5 and 5x6 mm.

*Summary of these 20 canals:*

In 4 canals the atrophy was secondary to lues.
Nasal infection in 2. Dental infection in 2.
In 2 the atrophy was secondary to pituitary disease.
Nasal obstruction.
In 2 the atrophy was secondary to multiple sclerosis.
Nose and throat negative.
In 2 the atrophy was secondary to cerebral hemorrhage.
Infected teeth and tonsils.
In 2 the atrophy was secondary to retinitis pigmentosa.
Slight nasal obstruction.

In 2 the atrophy was secondary to oxycephaly.
Nasal obstruction.
In 2 the atrophy was congenital.
Nasal obstruction.
In 4 (unilateral) the atrophy was secondary to retrobulbar neuritis probably of dental origin.
Nasal obstruction in 2. Suspicious tonsils in 3.

*Comment:*

There were but four cases in this series where the atrophy seemed to be secondary to retrobulbar neuritis and in three of these at least it appeared to be of dental origin. In most of the other cases the pathology found in the nose and throat did not appear to be a factor in the atrophy.

GROUP V

These canals are above normal, eleven in all, the measurements being 5x7, 5.5x6, 5.5x6.5, 6, 6x7 and 7 mm.

*Summary of these 11 canals:*

In 6 the atrophy was secondary to brain tumor.
Nose and throat negative in 4. Diseased tonsils and nasal obstruction in 2.
In 4 the atrophy was secondary to oxycephaly.
Nose and throat negative in all.
In 1 the atrophy was secondary to pituitary disease.
Nasal obstruction and suspicious tonsils.

*Comment:*

Associated with three of these eleven canals there were diseased tonsils and nasal obstruction. Atrophy in all, however, was due to the intracranial disease.

SUMMARY OF GROUPS I, II, III, IV, V

Nasal infection was responsible for the atrophy in twelve out of the 124 canals, or 10%. This occurred in the small canals,—nine in Group I and three in Group II. In four canals the atrophy was thought to be secondary to tonsilar infection. The other causes were as follows:—

In 24 canals the atrophy followed brain tumors or pituitary disease.
In 18 canals the atrophy was of luetic origin.
In 18 canals the atrophy was congenital.
In 14 canals the atrophy was from diseased teeth.
In 8 canals the atrophy followed retinitis pigmentosa.
In 6 canals the atrophy was from oxycephaly.
In 4 canals the atrophy was secondary to tonsilar infection.
In 4 canals the atrophy was from spinal meningitis.
In 4 canals the atrophy was from infantile paralysis.
In 4 canals the atrophy was from multiple sclerosis.
In 2 canals the atrophy followed cerebral hemorrhage.
In 2 canals the atrophy was unclassified.

LARGE OPTIC CANALS

In all the canals above normal there was intracranial involvement, i. e., there were six canals in brain tumor cases, one in a pituitary and four in tower skulls. The evidence from these few cases, therefore, indicates that increased intracranial pressure may produce a dilatation of the optic canals. The cause of this dilatation is probably the continuous pressure of the spinal fluid within the optic nerve sheath against the walls of the canal, this producing absorption of the bone.

Intracranial lesions were responsible for the atrophy in but 16% of the canals below normal.

When we contrast this 16% with 100% in the abnormally large canals we have facts difficult of interpretation unless we concede that increased intracranial pressure produces dilatation of the canals.

Of the twenty canals of normal size the atrophy in two was due to pituitary disease and in two to oxycephaly. In neither of these cases did the intracranial pressure seem to be as marked as in the cases with abnormally large canals, this opinion being based on the smaller size of the sella turcica, as compared with the sellae in the larger canal cases.

As previously stated, 16% of the cases with canals below normal had intracranial lesions. This shows that brain tumors do not necessarily produce dilatation of the canals. It is probable that the papilledema was either of short duration or absent, for according to Gowers and Barker papilledema is not present in from 10 to 20% of brain tumor cases.

SMALL OPTIC CANALS

The average diameter of twenty-eight canals in cases with congenital atrophy was 4.5 mm. which is approximately the average in the entire series, so that as far as this datum goes early atrophy of the nerve does not account for the small canals. While small canals seem to be a distinct factor in the production of atrophy, early atrophy does not explain their diminished size.

The practical point in this study is the question of enlarging the optic canal should one of these small canal cases be seen before the atrophy is complete. The canal can be approached by three routes, i. e., the frontal way as advised by Schloffer, the orbital route as performed by Hildebrand and the lateral route, the method employed by Dandy. Van der Hoeve in a recent article on Optic Nerve Tumors says:—"Operations by the lateral way give a liberal view of the condition at the intracranial optic foramen and allow us to enlarge the optic canal. . . . This operation can be used if the canal is con-

siderably deformed or absolutely too narrow, i. e., in cases of optic nerve involvement, as tower skull, fractures, etc." He also adds that "the canal operation for preservation of the visual power should be performed only if both optic nerves are endangered in such a measure that absolute blindness is to be feared."

These operations on the optic canal are not devoid of danger; they have been but rarely performed and would require all the skill and dexterity that comes only through long training in neural surgery. While I have never seen a case in the stage when it could have been beneficial, there are probably several in this series where it might have prevented the atrophy.

## CONCLUSIONS

In many of these cases where the atrophy was secondary to various other conditions, there was nasal obstruction, infected sinuses, teeth or tonsils. This fact needs emphasis as it is only too common a failing among some rhinologists to attribute to pathology in their own province most of the optic nerve disturbances.

Whatever scientific value this paper may have will be found in the comparison between the sizes of the optic canals in these various groups, and the sizes from a large number of normal cases similarly grouped. Fortunately this comparison could be made as I had the data from the measurements of 300 canals in skulls at the Harvard Medical School.

24% of all the optic atrophy canals I was agreeably surprised to find was in Group I, these being the very small canals rarely, if ever, found with normal vision, while in the Medical School series, which should represent the normal, there was 1% in Group I.

There can be little doubt that a proportion so marked as 24 to 1 can be interpreted in any other way than that the predisposition to permanent loss of vision is markedly increased when any process capable of producing atrophy, occurs within these abnormally small canals.

33% of the canals was in Group II, as contrasted with 7% found in the Medical School measurements. Here again with a proportion of 7 to 33 the diminished area of these canals contributes without doubt to the high percentage of atrophy, although not to the same extent as in Group I.

34% of the optic canals was in Groups III and IV, these being the normal canals and those but slightly below normal. According to the Medical School series there should have been 85% of all the canals in these groups. This clearly indicates that the canals as they approach the normal size rapidly cease to be a menace to their neural contents. Immunity to atrophy, however, is not found in any sized canals.

In the canals above normal (Group V) it was interesting to find how closely the two series compared. Thus, there should have been 7% according to the Medical School series, while as a matter of fact there was 8% in the optic atrophy group. This seems to indicate that in a certain small percentage of cases the size of the canal is not a factor in the atrophy. In these particular cases it was due to increased intracranial pressure.

## SUMMARY

This is the third paper devoted to the study of optic canals. In the first, it was shown that excessive pneumatization of the sinuses adjacent to the canals produced narrowing and distortion. In a series of radiographs it was found that patients with optic nerve disturbances had abnormally small canals. By analyzing thirty-six cases of optic nerve involvement it was found that in the smaller canals the vision in 50% was permanently impaired, while in patients with larger canals the percentage was much less.

Sixty-seven cases of optic atrophy were studied. These were divided into five groups according to the size of the optic canal. Nasal infection was responsible for atrophy in twelve out of one hundred and twenty-four canals, or 10%. This occurred in the small canals,—nine in Group I and three in Group II. The other causes were as follows:

In 24 canals the atrophy followed brain tumors or pituitary disease.
In 18 canals the atrophy was of luetic origin.
In 18 canals the atrophy was congenital.
In 14 canals the atrophy followed from diseased teeth.
In 8 canals the atrophy followed retinitic pigmentosa.
In 6 canals the atrophy was from oxycephaly.
In 4 canals the atrophy was secondary to tonsilar infection.
In 4 canals the atrophy was from spinal meningitis.
In 4 canals the atrophy was from infantile paralysis.
In 4 canals the atrophy was from multiple sclerosis.
In 2 canals the atrophy followed cerebral hemorrhage.
In 2 canals the atrophy was unclassified.

Increased intracranial pressure seemed to be responsible for the abnormally large optic canals as intracranial lesions were found in all. Atrophy of the optic nerve in infancy was not responsible for the very small canals.

While it is possible to surgically enlarge the optic canal, cases are rarely seen at the stage when it would be beneficial.

In comparing sizes of optic canals in these atrophy cases with the normals, it was found that in Group I the proportion was 24 to 1, so there would seem little doubt that a proportion so marked as this could be interpreted in any other way than that the predisposition to permanent loss of vision is markedly increased whenever any process capable of producing

atrophy occurs in abnormally small canals. As the canals approach normal size they rapidly cease to menace their neural contents, although atrophy may occur in canals of all sizes.

BIBLIOGRAPHY

White, Leon E.: An Anatomical and X-ray Study of the Optic Canals in Cases of Optic Nerve Involvement. Trans. Am. L., R. and O. Soc., 1923.

The Treatment of Optic Nerve Involvements as Determined by Optic Canal Radiographs. Trans. Am. Acad. Ophthal. and Oto-Laryng., 1923.

Schloffer: Klin. M. f. Augenh., 1913, Vol. 41, p. 1.

Hildebrand: Arch. f. clin. Chir., Vol. 124, p. 199.

Dandy: Am. Jour. of Ophth., 1922, Vol. 5, p. 169.

van der Hoeve, J.: Roentgenography of the Optic Foramen in Tumors and Diseases of the Optic Nerve. Am. Jour. of Ophthal., 1925, Vol. 8, No. 2,

Gowers, Sir William: Quoted by Osler, The Principles and Practice of Medicine. D. Appleton & Co., 1910.

Barker, L. F.: The Clinical Diagnosis of Internal Diseases, Vol. 3, p. 565. D. Appleton & Co., N. Y., 1916.

# MEDICAL PROGRESS

## PROGRESS IN SURGERY—1924

BY JOHN S. HODGSON, M.D.

Perthes, G.: Is Nerve Anastomosis or Myoplasty to be Preferred in the Treatment of Incurable Paralyses of the Facial Nerve? (*Zentralbl. f. Chir., li* 2073, 1924).

The author discusses the relative value of nerve anastomosis and myoplasty in incurable paralysis of the facial nerve. He cites one case in which the facial nerve had been destroyed by a middle ear operation. After six months an anastomosis was performed between the hypoglossal and facial nerves. Eleven years later the complete facial paralysis persisted. There were slight fibrillary twitchings and uncoördinated movements in the paralyzed side of the face accompanying deglutition or tongue movements. This slight gain was vitiated by a unilateral atrophy of the tongue, with difficulty in speech, and involvement of the tongue. A plastic operation by Lexer's method, consisting in the transplantation of the anterior portion of the masseter to the mimic musculature at the angle of the mouth, was performed. This corrected the salivation. In the second case, that of a girl of nineteen, with complete facial paralysis and reaction of degeneration, a strip of temporal muscle was implanted into the external canthus of the eye. Later a portion of the masseter was implanted into the angle of the mouth. After a few months the palpebral fissure could be closed at will by temporal muscle contraction, while the drooping of the angle of the mouth disappeared, and the mouth could be moved sidewise.

Davis, L. E., and Cushing, Harvey: Experiences with Blood Replacement during or after Intracranial Operations (*Surg., Gyn., and Obstet.*, XL, March, '25).

The authors describe a method of collecting and replacing blood lost during an operation. They employ a simple suction device by means of which blood is constantly aspirated from the field of operation through a glass nozzle and collected in a flask surrounded by warm water. The blood is kept liquid by frequent additions of 2% sodium citrate solution. Pledgets saturated with blood during the operation are wrung out, and this fluid and blood is also used. Before its reinfusion the blood is twice filtered through many layers of gauze. Blood pressure is carefully recorded during the entire operation, and reinfusion is performed whenever indicated, either during or following operation. There usually results a favorable pressor effect blood pressure rising and pulse slowing. This rise in pressure is well sustained and is considered life saving in many instances. Untoward effects obtained from the usual methods of transfusion are largely eliminated, blood grouping is not required, and the complication of a donor is obviated. There is cited the case of a degenerated glioma of the posterior hemisphere previously operated upon and treated by X-ray, in which a considerable depressor effect, nausea and sweating occurred. Possibly the juice from disorganized tissue following irradiation, mixed with the blood reinfused, was the cause of the reaction. It is admitted that whole blood transfusion is preferable and necessary in certain cases, but it is felt that this method is of distinct value, reduces the number of necessary two-stage operations, and is at times life saving. The method has been in use a year; its field of usefulness in other than intracranial surgery is indicated.

Mayo, W. J.: The Surgical Treatment of Hepatic Cirrhoses (*Ann. Surg.*, 80, 1924).

The author calls attention to the parallelism existing in the relation of an enlarged spleen to both portal and biliary cirrhosis. Cirrhoses of the liver are divided into two groups: (1) portal cirrhosis, the result of deposits of connective tissue around the radicles of the portal vein, causing ascites and hemorrhages from the stomach, and (2) biliary cirrhosis, the result of deposits of connective tissue around the biliary duct system, causing chronic jaundice. Portal cirrhosis may be gastrointestinal in origin, in which the spleen is not greatly enlarged. In suitable cases splenectomy and the Talma-Morison operation combined may be of value. Splenectomy in addition to the Talma-Morison operation, however, carries increased risk, and should not be performed until the functional capacity of the liver has been tested.

Investigation at the Mayo Clinic has shown that when the liver function is reduced to below 25%, any serious operation will probably end fatally. In the secondary splenic type of portal cirrhosis, occurring in the late, Banti stage, of splenic anemia, splenectomy gives splendid results. The common forms of biliary cirrhosis are the result of obstruction and infection of the biliary ducts, usually associated with gall stones, and removal of the obstruction or infection usually effects a cure. The spleen is not greatly enlarged in such cases and splenectomy appears unnecessary. In the splenic type of biliary cirrhosis, however, very chronic in character, splenomegalia is a prominent symptom, and splenectomy may be indicated.

McNee, J. W., Van den Bergh, H., Willcox, Sir W., and others (*Brit. M. J.*, II, 495, 1924).

Van den Bergh has developed a delicate method by which small amounts of bilirubin can be detected and measured quantitatively in small amounts of albuminous fluids such as blood serum, and qualitative differences in the bilirubin present in the serum in different clinical forms of jaundice can be determined readily.

Mann of the Mayo Clinic has devised a new technique for total extirpation of the liver in dogs. In these animals Mann found that bile pigment appeared in the blood serum and urine within a few hours after the liver had been removed. This suggests that a true hemolytic jaundice has been produced experimentally.

Gonzalez, A., and Karr, W. G.: A Comparative Study of the Phenoltetrachlorphthalein and Hemoclastic Tests of Liver Function (*Arch. Int. Med.*, XXXIV, 282, 1924).

The hemoclastic reaction measures the physiological capacity of the liver cells directly. In the dye test this measurement is dependent upon the patency of the biliary passages. The hemoclastic reaction seems to be more sensitive than the dye test, but a combination of the two is often helpful in the diagnosis and prognosis of obscure hepatic disease. Emphasis is laid on the large factor of safety in the liver, which precludes the use of any functional test to determine the presence of a minor hepatic disturbance.

Halpert, B.: New Methods of Studying the Gall Bladder (Neue Wege in der Gallenblasenforschung) (*Med. Klin.*, XX, 408, 1924).

On the basis of numerous clinical and anatomical investigations the author comes to the conclusion that the cystic duct should be considered only as an afferent duct to the gall bladder. The arrangement of Heister's valves is such that they favor the access of bile to the gall bladder and prevent or hinder its reflux by the same route. The efferent passages are normally the lymphatics and blood vessels. The healthy mucosa of the gall bladder is believed to resorb the normal secretion of the liver completely. In the gall bladder the bile becomes thickened, and the substances become resorbed. It is the function of the mucosa of the gall bladder to preserve the valuable secretion of the liver for the bodily economy during the intervals of digestion; it breaks down the bile into its elements, and, conducting them by way of its lymphatics and blood vessels, places them again at the disposal of the body. If for any reason resorption does not take place, stasis of the bile results, at first in the gall bladder, and later also in the bile ducts.

Carman, R. D., and Counseller, V. S.: The Roentgenological Diagnosis of Cholecystic Disease with the Aid of the Sodium Salt of Tetrabromphenolphthalein (*Am. Jour. Roentgenol.*, XII, 403, 1924).

The administration of sodium tetrabromphenolphthalein was found to be inadvisable when obstruction of the biliary passages was known to be present. The method was found to be unsuitable also for patients with any type of cardio-vascular disease, particularly those with arterio-sclerosis and chronic cardiac disease. The typical severe reaction was one of vasomotor shock, beginning usually within five to ten minutes after the injection of the dye. There was usually a rise in the blood pressure, followed in from three to five minutes by a sharp fall. Gradually during the next fifteen to twenty minutes the pressure returned to its previous level. Because of the numerous reactions, the standard dose was reduced from 5.5 to 4.5 grams. Even with this dose there were occasionally moderate reactions. There were also delayed reactions developing four to five hours after the injection. Tissue taken from the liver at operation revealed no changes attributable to the dye. In the normal response the gall bladder will retain sufficient dye to cast a shadow at the fourth or fifth hour after the injection. This shadow attains its greatest density some time between the eighth and 24th hours, becomes progressively fainter thereafter, and should disappear between the 24th and 48th hours. The shadow is oval or pyriform, with an even contour, and should be homogeneous. It is larger at earlier than succeeding hours. Abnormal responses include failure of the gall bladder to fill with the dye, scanty filling, delayed filling, marked delay of emptying, deformity of contour, unvarying size of the shadow, constant extremes of size, and mottling or central filling defects. Of 178 patients examined by the Graham method, 39 were operated upon. Roentgenograms were made at 5, 8, and 24 hours after the administration of the dye. The gall bladder failed to fill with the dye in 19 of 25 cases of gall stones, and in only two cases could the findings be construed as negative. In the remaining six cases dye entered the gall bladder but two had a mottled shadow, one showed dense shadows of

stones within that of the gall bladder, and the other was faintly visible at the eighth hour only. Of 14 cases without stones, the gall bladder failed to fill with the dye in eight. In three cases abnormalities of filling were noted, and in the remaining three no abnormalities of filling were observed.

Graham, Cole, and Copher. Roentgenological Visualization of the Gall Bladder by the Intravenous Injection of Tetrabromphenolphthalein (*Ann. Surg.*, 80, 473, 1924).

The calcium salt of tetrabromphenolphthalein has been used but discarded because of its relative insolubility, requiring large amounts of fluid, and the unpleasant effects, as dizziness, headache, nausea and vomiting. Therefore the sodium salt has been used. The stomach should be empty at the time of injection. Five to 5.5 grams of the sodium salt are dissolved in 35 to 40 c.c. of water. Aside from its value in the diagnosis of certain abnormalities of the biliary tract, the method may prove of value as a test of the functional capacity of the liver, as judged, for example, by the time required for the appearance of the shadow. The method will permit of the addition of new facts concerning the normal physiology of the gall bladder, especially as regards the question of emptying time, etc. Visualization requires (1) that the functional capacity of the liver be sufficient to permit it to secrete the substance in the bile, (2) that the cystic duct be open to permit the substance to enter the gall bladder, and (3) that the concentrating function of the gall bladder be sufficiently good to permit concentration of the substance within the gall bladder. Theoretically, therefore, the best shadows should be obtained in normal gall bladders, and a failure to obtain any shadow at all, would almost certainly denote serious pathology of the biliary tract, including the gall bladder. The writers feel that failure to obtain a definite shadow probably indicates a high grade cholecystitis. Gall stones have been diagnosed in several instances, as shown by their casting less opaque shadows than that of the injected substance. Adhesions have been recognized by the abnormal contour of the wall of the gall bladder. To insure the best results a series of plates over 36 hours must be taken. After 4 to 7 hours from the time of injection a faint but definite outline of the gall bladder appears, which is seen to have the contour of the normal gall bladder, but to be somewhat larger than normal gall bladders usually seen at laparotomy. At the end of 24 hours the shadow is much more distinct but contracted down to about one half its earlier size. From then on until about the forty-eighth hour the shadow diminishes in size and fades gradually. In cases of simple cholecystitis without stones or adhesions the appearance of the shadow may be delayed, and it may be much less dense than the normal. Injection of the sodium salt intravenously is given in 2 doses half an hour apart before breakfast. 5.5 grams are dissolved in 40 c.c. of distilled water. Great care must be taken to prevent extravasation of the solution into the tissues of the arm.

Orders for the patient:—

1. Omit breakfast.
2. Omit lunch (may have glass of milk).
3. Lie on right side of abdomen or be up walking around.
4. Omit proteins from evening meal.
5. Sodium bicarbonate, gr. xl, every 3 hours for 48 hours.

X-rays are taken at 4, 8, 24 and 32 hours. The only toxic effects noted have been dizziness, nausea, and vomiting.

Whitaker, L. R,, and Milliken, G.: A Comparison of Sodium Tetrabromphenolphthalein with Sodium Tetraiodophenolphthalein in Gall Bladder Radiography (*Surg., Gyn., and Obstet.*, XL, 17, Jan., 1925).

Graham and Cole devised the method of roentgenologic visualization of the gall bladder. They first advocated calcium tetrabromphenolphthalein and more recently sodium tetrabromphenolphthalein. Whitaker and Milliken believe that sodium tetraiodophenolphthalein has definite advantages over the analogous bromine salt for use in biliary cystography. Elements are opaque to X-rays approximately in proportion to their atomic weights. The atomic weight of bromine is 80, that of iodine 127. The toxicity of the bromine and iodine salts is about equal, but as the dose of the iodine salt necessary to produce a satisfactory shadow is only one half that of the bromine salt there is a wide margin of safety in favor of the iodine compound. Toxicity experiments were conducted on animals, and one of the authors submitted to the test first with the bromine and later with the iodine salt given intravenously. With the bromine salt there was slight pain in the injected vein and a slight initial rise in pulse and respiration. This returned to normal in 2 to 3 hours. There was slight headache, considerable lumbar pain and some drowsiness until 6 hours after injection, when nausea and abdominal cramps developed. There was vomiting of water and mucus and later bile. Vomiting continued for about 2 hours. There was a soft free bowel movement. The following day the subject felt better though weak and tired, and had considerable soreness in the lower back. He had recovered fully after 2 days. With the iodine salt there was only very slight rise in pulse. All other symptoms following the use of the bromine salt were absent except slight weakness the night of injection, and soreness in the back the next morning. The dose of the bromine salt was 0.08 gram per kilogram of body weight, that of the iodine salt 0.05 gram per kilogram. The denser shadow was cast by the iodine salt.

Milliken and Whitaker: The Clinical Use of Sodium Tetraiodophenolphthalein in Cholecystography (*Surg., Gen., and Obstet.*, XL, 646, May, 1925).

The authors now find that 0.040 gram per kilogram of body weight gives satisfactory shadows and causes negligible reactions. The quantity of salt needed is weighed and dissolved in 100 c.c. of freshly distilled water; a 10% solution is made. 1.5 to 2 c.c. of a 10% solution of sodium carbonate is added to keep the salt in solution. The solution is autoclaved at 15 lbs. for 30 minutes and is never used after 24 hours. The injection is made in the morning. As active digestion is contraindicated no food should be taken during the first 9 hours, though water is allowed. After the injection the patient should remain in bed, preferably upon his right side, though he may sit up or get up if he desires. Roentgenograms are made at 9, 12, 24, and 36 hour intervals. The drug has been given in cases of myocarditis and slight decompensation without reaction. The use of the drug is contraindicated in intense jaundice as from common bile duct obstruction because only ill defined gall bladder shadows are obtained. Injection of the drug under these conditions would probably not be attended by any great danger because in dogs and cats ligation of the common bile duct increases the toxicity only 25%. The authors conclude that the test may be used without risk in any patient whose general condition would permit operative intervention. The diagnosis of pathological conditions of the gall bladder by means of cholecystography has been found to be correct in 95% of the series of proved cases, while the clinical impression was correct in only 65%.

Whitaker, Milliken, and Vogt: The Oral Administration of Sodium Tetraiodophenolphthalein for Cholecystography (*Surg., Gyn., and Obstet.*, XL, June, 1925).

The authors have devised pills of sodium tetraiodophenolphthalein double coated with salol in syrup of tolu. Definite gall bladder shadows were produced in 93% of the normal subjects and 65% of the patients. The absence of a shadow by the intravenous method has meant a pathological condition of the gall bladder in 100% of their proved cases. On the whole the shadows produced by the oral method were less dense than those by the intravenous method with the iodine salt; this fact indicates the need of good roentgenographic technique, especially since the absence of a shadow under these conditions means cholecystic disease. An illustration of the difference in reaction between the bromine and iodine salt injection and the oral administration of the iodine salt is shown in a case in which after bromine injection the patient vomited and was quite ill, after the iodine injection he felt sleepy and somewhat weak, and after the iodine salt by mouth had no symptoms. The patient does not need to be hospitalized for this test and can go about his usual activities. Five grain salol coated pills to the amount of 0.08 gram per kilogram of body weight, from 3.5 to 6.5 grams, are issued. Not more than 20 pills are given. A light supper of bread and tea, coffee, cocoa or milk is allowed, and starting at 8 or 9 o'clock, p. m., 4 pills are taken every 15 minutes with half a glass of water until the whole number is taken. The patient lies in bed on his right side during the taking of the pills and for an hour or more thereafter. The next morning the patient is X-rayed 12 and 15 hours after taking the pills, no food being eaten meanwhile. After the 15-hour film the patient eats a meal, and an hour afterward more roentgenograms are made to see if the gall bladder shrinks down during digestion, thus indicating normal contractility. Of 44 persons tested 27 had no symptoms, 5 vomited 1 to 3 times, 5 had mild diarrhea, and 7 had slight nausea. The symptoms were less severe than those sometimes following intravenous injection. Vomiting with the oral method is probably due to gastric irritation by an improperly coated pill or to the salol coating, and with the intravenous method is probably toxic. The authors give a dose of magnesium sulphate following the last roentgenograms to neutralize any phenol resulting from the decomposition of salol and to remove undecomposed salol. The authors believe that the oral method of administration has several important advantages in that it is convenient both for patient and physician, that it can be used by any roentgenologist in his office, and that in general it gives fewer troublesome symptoms. The intravenous method, on the other hand, gives somewhat better shadows, is better controlled and perhaps more dependable. They believe in first giving the drug by mouth. If a normal shadow is obtained the probability of gall bladder disease is slight. If the shadow is imperfect or absent then an intravenous injection of the drug should be given to confirm the findings. Results by the intravenous method have yielded correct diagnoses in 95% of proved cases.

McVicar, C. S.: A Discussion of the Clinical and Laboratory Findings in Certain Cases of Obstruction in the Upper Gastro Intestinal Tract; the Rôle of the Blood Chemistry in the Diagnosis, Prognosis, and Treatment of this condition (*Am. J. Med. Sc., cl,* XIX, 224, 1925).

The author emphasizes the value of frequent chemical examinations of the blood in the determination of progress in the treatment of organic obstruction prior to operation and in post operative ileus. The signs suggesting treatment are dehydration, diminished output of urine, a low blood pressure, shock-like prostration, a high non-protein content of the blood, low blood

chlorides, and a tendency toward a high carbon dioxide combining power of the plasma. The administration of water is indicated to combat dehydration, to counteract shock, to promote diuresis, and to get rid of the nitrogenous waste products. It is not clear whether the high blood urea is indicative of excessive tissue catabolism or of retention due to renal insufficiency or both. Renal insufficiency is a factor. Sugar has been used to spare protein and intravenously to promote diuresis. The experiments of MacCallum and experience of the Mayo clinic indicate the desirability of administering salt. There seems to be a markedly increased demand of the tissues for sodium chlorid. Blood chlorides are lowered. It has been suggested that this is due to loss of HCl from the gastric juice by vomiting. However, in pyloric occlusion in rabbits that cannot vomit Haden and Orr have noted a sharp fall in the chlorides. They have also observed a fall in the chlorides after obstruction of the cardia. Since it is impracticable clinically to administer HCl in amounts adequate to replace the amount lost by vomiting or lavage, sodium chloride has been used and found efficient. Lavage is practiced on the theory that the secretion contains a toxin. In functional ileus lavage removes a weight which would otherwise interfere with the recovery of tone. Another and possibly the most important fact disclosed by blood chemistry is that in this type of toxemia there is an invariable tendency toward alkalosis. This therefore clearly contraindicates the use of alkalis in proved or suspected cases of toxemia due to organic or functional obstruction in the upper gastro-intestinal tract. With the toxemia of high intestinal obstruction, characteristic changes occur in the blood, namely a rise in the blood urea, a fall in the plasma chlorides, and a rise in the carbon dioxide combining power of the plasma. By a study of the chemistry of the blood the condition can be recognized early, its severity can be measured, and the progress of the treatment can be watched.

Youmans, J. B., and Greene, I. W.: Gastric Tetany (*J. A. M. A.*, 84, 808, Mar. 14, 1925).

As shown by the experimental work of McCann, and of MacCallum and his associates, gastric tetany is accompanied by and associated with an alkalosis, as indicated by an increase in the blood bicarborate and a decrease in the hydrogen ion concentration. There also occurs a lowered concentration of blood and urinary chlorides, an increase in the phosphates and sulphates, and a variable rise in the non-protein nitrogen of the blood. In addition, under similar conditions, blood changes in the same direction have been noted in animals and in patients exhibiting to a less degree some of the signs and symptoms observed in tetany. That these changes in the constitution of the blood are due to the loss of HCl by vomiting or gastric lavage seems definitely established, but the exact nature of the mechanism responsible for the production of the tetanic phenomena remains in doubt. Among the explanations of the production of gastric tetany have been (1) the direct effect of the increased alkalinity on the peripheral nerves causing increased excitability. This possibility is supported by certain fundamental experiments of the type reported by McClendon, and indirectly by the work of Grant and Goldman on the tetany of hyperpnea. (2) An indirect action of the increased alkalinity by way of an anoxemia and consequent increase in nerve irritability (*Morris, Brit. M. J.*, 1922). (3) A disturbance in the concentration and ratios of various cations of the body resulting from the change in acid-base balance. (4) A disturbance in the anion concentrations and ratios not necessarily caused by the altered acid-base balance itself, but accompanying it and due to the same cause, i. e., to the loss of HCl. This explanation involves particularly the bicarbonate, phosphate and chlorine ions, and includes the possibility of the effect of disturbed relations between anion and cation. As MacCallum suggests, while the final change in the blood that produces the symptoms of tetany is the same for all types, the changes occurring before this point is reached may vary greatly in the different forms, and the road to tetany of the gastric type may, over a large part of its course, be vastly different from that of the other varieties. Despite the uncertainty as to the exact mechanism of tetany production, the fact remains that gastric tetany is accompanied by an alkalosis, and recovery from the attack is associated with a restoration to normal acid-base equilibrium. The possibility of successfully treating this condition by replacing missing acid, thereby restoring the acid-base balance to normal, was therefore apparent. McCann reported the relief of symptoms in dogs with artificial pyloric obstruction by administering HCl by the duodenum. MacCallum, using smaller doses intravenously, failed to corroborate this finding, but he was partly successful in preventing attacks of tetany in dogs with experimental pyloric obstruction by injecting large amounts of sodium chloride solution. Sodium chloride in large doses has been used experimentally and clinically by Haden and Orr, and clinically by Dixon, and successful results have been reported. Haden and Orr feel that the decrease in chloride concentration is the important factor in this condition. In 1922 McCann gave a solution of ammonium chloride intravenously to a woman suffering from an attack of gastric tetany. He reported rapid and complete recovery from the attack. Recently Ellis reported the successful use of ammonium chloride by rectum in two cases of gastric tetany. The authors add the report of a case

of gastric tetany successfully treated by the injection of ammonium chloride intravenously. There is no intention to replace surgical treatment for the removal of the underlying cause of the condition, i. e., pyloric obstruction. It is believed, however, that this treatment will so improve the preoperative condition that surgical treatment, heretofore withheld because of the apparent hopelessness of the condition, will be more often used. The case reported is that of a woman of 50 with signs of high obstruction, acetone and diacetic acid in the urine, typical carpopedal spasm and facial twitching. The patient was stuporous and gave a typical reaction of tetany, i. e., cathodal closure tetany=less than 1 milliampere. The $CO_2$ combining power of the blood as 100+% by volume. The whole blood chlorides were 370 mgm. per 100 c.c. The respirations were slow and very shallow. 400 c.c. of 0.82% solution of ammonium chloride was given intravenously after testing for possible hemolysis. In the course of 4 hours all symptoms greatly improved, the face became flushed, respirations rose to 18 and there was diminution and later disappearance of the spasm. Two days later operation was performed. Pyloric obstruction due to old post operative adhesions was found. A posterior gastro-enterostomy was done. Following operation some spasm of the hands was noted. This promptly disappeared with the injection intravenosly of 400 c.c. of 0.82% ammonium chloride. Murray has suggested that in cases of obstruction, the degree of alkalosis and the development of tetany are dependent on the proportion of alkali (intestinal contents) to acid (gastric contents) lost. He likewise suggests that this relationship may be of assistance in determining the location of an obstruction, greater increases in the $CO_2$ combining power being present the nearer the obstruction is to the pylorus. The provisional diagnosis of acidosis at the time of admission is of interest in view of the subsequent finding of alkalosis and the development of tetany. Ellis has recently reported the presence of a ketonuria in cases of alkalosis, and he suggests that it is due to a deficient oxidation in the tissues, the result of anoxemia. The importance of the possible presence of a ketonuria lies in the danger of a mistaken diagnosis and consequent treatment of a sort to increase the alkalosis. Such a possibility emphasizes the importance of determinations of the $CO_2$ capacity or $P_H$ of the blood in cases of frequent and persistent vomiting, especially if an obstruction is suspected. Attention should be drawn to the possible ill effects of certain common methods of treatment that might be used in cases of gastric tetany. Gastric lavage, as frequently recommended is contraindicated in that it removes more acid from the stomach and therefore increases the alkalosis. It has, for example, been noted that attacks of tetany seem to have been initiated by gastric lavage. The common tendency to administer alkalis in view of the presence at times of a ketonuria may likewise increase the alkalosis.

Portis and Portis: Experimental High Intestinal Obstruction. Relief by Irrigation and Control of Alkalosis. (*J. A. M. A.* 85-574, Aug. 22, '25.)

The severe reaction following high intestinal obstruction is due to some highly toxic substance. Whipple and his co-workers consider the poison to be a primary proteose formed by perverted activity of the intestina mucosa. Dragstedt and his associates attach great importance to the action of bacteria, both proteolytic and putrefactive. Draper believes that there is a perverted activity of the mucosal epithelium. Davis and Stone have shown that loop washings can become toxic outside the body and produce symptoms like the contents of closed loops when injected into animals. They therefore conclude that a perverted activity of the mucosa is not present. Histamin has been isolated by some workers and they have considered this important in the production of the symptoms. Hausler and Foster stated that in high intestinal obstruction a shock complex is more of a factor than a toxic reaction. It seems that the toxic products formed in high intestinal obstruction are probably due to bacteria as well as to an abnormal function of the duodenal mucosa. Haden and Orr observed in a number of patients following gastro-enterostomy that more or less serious symptoms arose, attributed by them to absorption of a poison formed by stasis in the duodenum. Under such conditions there is a marked rise of non protein nitrogen bodies in the blood, with marked increase in the nitrogen excretion from the kidney. There is a low chloride content in the blood and there may be a suppression in the urine. Sodium is thus left free and unites with the carbonates to give sodium carbonate and eventually an alkalosis. Dixon believes that the marked depression of chlorides in the blood plasma may be due to a union of the toxins with the chlorides. High intestinal obstruction is usually accompanied by a marked increase in the $CO_2$ combining power. This tendency to alkalosis may explain the gastric tetany observed in experimental animals and is probably a real factor in a fatal termination. The authors used 18 dogs and performed their experiments as follows: A gastroenterostomy was done in all cases. In the first series gastroenterostomy alone was done, in the second series pyloric occlusion was added, and in the third series pyloric occlusion and a constricting rubber band was placed on the proximal loop near the gastroenterostomy opening. In this manner a positive obstruction was produced with the duodenum forming a closed loop. In the earlier animals, in the absence of irrigation, death occurred in the first 24 hours. The contents of the

obstructed proximal loop were highly toxic, as from 0.5 to 1 c.c. of the Berkefeld filtrate was sufficient to kill a normal guinea pig in 12 to 24 hours after intraperitoneal injection. Bacteriologic cultures showed only the constant presence of B. coli and some gram positive cocci. $CO_2$ determinations by the Van Slyke method were made on the last few dogs. In the later work 2 tubes were inserted into the proximal duodenal loop, one to the closed end, and the other to near the gastroenterostomy opening. Physiologic sodium chloride or Ringer's solution was introduced through the longer tube at a slow constant rate from an elevated bottle. The fluid was withdrawn by siphonage by means of a shorter tube. A third tube was left in the stomach for washing out gastric secretion, and for the later introduction of fluids and food. Irrigation was carried on continuously for 24 hours and for a short time after the occluding band near the gastroenterostomy stoma had been removed. The band occluding the jejunum was removed after 24 hours. 8000 c.c. of physiologic NaCl solution was passed into the duodenal loop and 7930 c.c. of solution returned. Muscular twitchings developed shortly after the operation These became exaggerated, and 25 hours after operation the dog developed severe generalized convulsions and did not respond to external stimuli. The $CO_2$ combining power at this time showed 100, or almost twice normal. After the administration of 120 c.c. of 0.3% HCl the dog underwent a remarkable change, first regaining consciousness, later reacting to external stimuli, and finally seeming quite well. A second $CO_2$ combining power taken ½ hour after the administration of acid showed it to be 73, a decrease of one fourth. The duodenum was then irrigated during the next 7 hours with 2100 c.c. of salt solution, but only 1700 c.c. was returned. Evidently the 400 c.c. had passed on by the now free duodenal loop. The remainder of the day 0.3% HCl was given, as was indicated, and the $CO_2$ combining power was reduced to 66 and finally 56. The general condition of the dog constantly improved, and after 48 hours the dog was free from twitchings and tetany and walked about and acted normally and has remained normal since.

## LAWS RELATING TO MARRIAGE IN NORWAY

It is of interest to note that the relationship of health to marriage has lately been stressed in a practical manner in Norway. From documents kindly provided by Fru Scheel of the Norwegian Red Cross, it appears that on January 1st, 1919, a new Marriage Law came into force.

An insane person may not marry, and a person suffering from syphilis in an infectious stage is also forbidden to marry. A person suffering from other forms of venereal disease which are still infectious, or from epilepsy or leprosy, must not marry unless the other party to the marriage is cognisant of these facts, and both parties have been verbally instructed by a doctor as to the dangers of the disease in question. Doctors are absolved from their duties of professional secrecy in this matter and must, indeed, notify the authorities if they are cognisant of the existence of any of the above-named diseases in a marriage candidate. If it is known that one of the prospective parties to a marriage has been insane, and has not been certified as cured, or if there are reasons for thinking that one or the other is at present insane, a certificate must be provided indicating that insanity is not at present demonstrable. A person who was insane at the time of marriage may claim dissolution thereof, and a divorce may also be obtained by the other party if he or she was in ignorance of the other party's insanity at the time of the marriage.

The following questions must be answered in writing by both parties:

(1) Are you related by birth or marriage to the other party in the circumstances referred to in Section 7 or Section 8 of the Marriage Law?

(2) Have you been married previously, and if so, to whom?

(3) Have you children born out of wedlock, and if so, how many?

(4) Are you suffering from:

(a) Syphilis which is still in an infectious stage.

(b) Other venereal disease which is still infectious, or epilepsy or leprosy?

The penalty for false statements may be up to two years imprisonment.—*The World's Health.*

## THE WORLD'S CHILDREN

### MONTANA SAVES MOTHERS AND BABIES

Lower infant and maternal mortality rates for Montana are shown in a health review published lately by the Montana Department of Health. In 1923 the infant death date was 71.8 and in 1924, 65.8. The maternal mortality rate dropped from 7.5 in 1923 to 7.0 in 1924.

### SWITZERLAND

Accident insurance for public school children is to be provided by the Government of Zurich, Switzerland, as has already been done in Bern and Bazelland. At present 150 communities in Zurich are providing this insurance paying an average of 1,000 francs in case of accidental death, 5,000 in case of disability, and 3 francs a day for medical treatment.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

## CASE 11431

### MEDICAL DEPARTMENT

An American forty-eight years old, a grocer's clerk, was sent from the Consultation Clinic July 27 for study of dyspnea and edema. He had been married eight years. His wife had had no pregnancies. He had always had very good health. For five years he had had mild headaches once in two weeks, usually in the afternoon, and for the same length of time he had had occasional nosebleed, especially when he was excited. He had occasional sore throat. A year before admission he had tonsillitis and occasional cough with bad colds. He had occasional cramps in the legs.

A little over three months before admission he noticed that he had lost a little vigor and tired more easily than before, especially his feet and legs. Two months before admission he found his feet were swollen at night. This swelling increased until at admission they were swollen in the morning. Two weeks after the onset of the edema his abdomen began to swell, at first below the umbilicus; later it seemed to fill up below the chest. Three weeks before admission he began to be short of breath and to have hacking cough, hoarseness and diarrhea. Three days later he had poor appetite, pain in the right lower chest and a yellow tint in the sclerae. The pain began in the right shoulder blade and worked around to the right hypochondriac region, was knife-like, occurred once or twice a day and lasted about half an hour. During the illness he had had two or three mild headaches coming on about dark and lasting two or three hours. July 25 he had a mild nosebleed. The dyspnea had grown progressively worse until for the past ten nights he had had to sit up part of the night because of orthopnea. He was unable to lie on his left side. He urinated once or twice at night. He had had some palpitation on exertion. The diarrhea had caused five stools daily and some discomfort from his hemorrhoids. For several days there had been a small spot on each hip which itched considerably. He had gained about twelve pounds during the illness. Ten days before admission he stopped work.

At the Consultation Clinic the chest expansion was greater on the left than on the right. Tactile fremitus was absent in the lower two-thirds. The voice sounds were egophonic. There were râles over this area. The left lung was negative. There were dilated veins over the abdomen. The weight was 223½ pounds.

Examination showed an obese man, dyspneic, with red face, slight cyanosis and a subicteric tint. The veins about the nose and cheeks were dilated. The sclerae were muddy and subicteric. There were bilateral pinguiculae. The upper teeth were false, the lower decayed and foul snags. The throat was injected. There were a few questionable small glands high in the axilla. The right chest was flat back and front from the apex to the base, with decreased voice and fremitus, more in front. On the left there was increased bronchovesicular breathing throughout with increased fremitus and voice sounds. There were râles at the left base in the anterior axillary line on deep inspiration, pleuritic in quality. The apex impulse of the heart was seen and felt in the fifth space 14 cm. to the left of midsternum, coinciding with the left border of dullness. The midclavicular line was 9 cm. to the left. The right border was not determined. The supracardiac dullness was 4 cm. to the left. The action was rapid and forceful. There were no murmurs. The pulmonic second sound was accentuated. The action was regular. The walls of the radial arteries were palpable. The blood pressure was 165/100. Electrocardiogram showed normal rhythm, rate 110, diaphasic T wave, slight left axis deviation. The abdomen was distended and tense. The liver dullness extended below the costal margin and to the left. Traube's space was dull. There was movable dullness in the flanks and questionable fluid wave. There were distended tortuous deep veins over the lower chest and upper abdomen. There was marked edema of the legs and ankles, edema of the prepuce and pigmentation over the lower third of the legs. There were a few external hemorrhoids.

The temperature was 97.7° to 99.9° with a terminal rise to 103.2°. The pulse was 84 to 132. The respirations were 14 to 30. The amount of urine was normal, the specific gravity 1.030 to 1.028, no albumin or sugar, rare red blood corpuscles at one of two examinations, 8 to 10 leucocytes per high power field. The renal function was 45 per cent. The hemoglobin was 70 per cent. at two examinations, the leucocytes 6,600, the polynuclears 75 per cent., the reds 4,824,000 to 4,856,000, the smear normal. A Wassermann was negative. The coagulation time was 7½ to 12 minutes. The clot retraction was poor at the end of three hours. The non-protein nitrogen was 47 mgm. The stools showed a positive guaiac at one of two examinations, but no macroscopic blood. An abdominal tap July 27 gave 700 c.c. of pale straw colored fluid, specific gravity 1.008, 807 cells, 83 per cent. lymphocytes, 17 per cent. polymorphonuclears, culture

sterile. A chest tap gave 3,360 c.c. of dark yellow fluid, specific gravity 1.012, 422 cells, 89 per cent. lymphocytes, 11 per cent. polymorphonuclears, culture sterile. July 30 an abdominal tap gave 35 c.c. of pale fluid, specific gravity 1.006, culture sterile, chlorids 669, protein 268 mgm. per 100 c.c., non-protein nitrogen 31, sugar 122. Plasma taken simultaneously showed non-protein nitrogen 34, chlorids 603, protein 5.995 grams per 100 c.c., sugar 130. A chest tap August 1 gave 2,850 c.c. of cloudy orange yellow fluid, specific gravity 1.010, 19,000 cells, 80 per cent. polymorphonuclears, 10 per cent. lymphocytes, 10 per cent. mononuclears, guaiac very strongly positive. Culture showed a profuse growth of colon-like bacilli. Guinea pig tests of the abdominal and chest fluids were negative. X-ray showed markedly increased density of the right side of the chest. The density was uniform and extended from the apex to the diaphragm, obliterating the outline of the diaphragm and the costophrenic angle. The cardiac shadow was displaced considerably to the left. The following day a second examination gave essentially the same findings as the first. Examination of the stomach in the recumbent position only showed no definite evidence of organic disease of the stomach or duodenum.

After the removal of the fluid no spleen could be felt and the sounds in the right chest persisted anteriorly higher than had been expected, including the bronchovesicular breathing and the coarse moist râles in the third and fourth right spaces. August 3 X-ray treatment of the chest and abdomen was given. That day the patient was slightly irrational. August 5 he died.

Shows markedly increased density of the right side of the chest, uniform and extending from the apex to the diaphragm, obliterating the outline of the diaphragm and the costophrenic angle. The cardiac shadow is displaced considerably to the left.

## Discussion

BY DR. RICHARD C. CABOT

### NOTES ON THE HISTORY

The question should come into everybody's mind: what are the possibilities and probabili-

ties when a person of this age, forty-eight, consults us for dyspnea and edema? Of course there are two which stand out ahead of anything else,—cardiac disease and renal disease. We can have dyspnea and edema from local pressure, from malignant disease, with a sound heart and sound kidneys. Also trichinosis and other rare diseases like that are in the background of our minds. But I should say the chances are ninety-nine out of a hundred that the case will turn out to be heart or kidney or both, although we do not know anything beyond the first two lines of the history.

"For five years he had had mild headaches." We say to ourselves, "Here is a man who begins to have headaches at forty-three." Why does a man begin to have headaches at that age? Ordinarily it is not eye-strain. Sometimes it is from syphilis, but it is more often from chronic nephritis than from any other one thing we can name. So that stays in the mind as a possibility linking with the other possibility, and to be further identified as the other facts in the case come out.

Nosebleed: if we can rule out local irritations and can prove that he did not scratch or otherwise traumatize his nose, that these were really spontaneous nosebleeds, they point to the same diagnosis as the other symptoms. Nosebleeds at forty-eight are more common in nephritis than in any other disease. They came especially when he was excited, probably when his blood pressure was high, so that he would be more likely to break a small vessel.

I cannot make anything out of the remaining symptoms. They do not point to anything that I know.

This case is very well written from the point of view of showing the march of symptoms: three months ago, loss of vigor; two months ago edema at night; two weeks after the onset of edema this thing creeps up until we begin to get the abdomen swelling and to have ascites.

The shortness of breath and cough of course naturally would be thought of, on the basis of the hypothesis that we started with, as passive congestion. Hoarseness we cannot so easily work into anything that I have been thinking of. If he had an aneurism of course that would account for his hoarseness. We do not have hoarseness from passive congestion. Diarrhea also does not work into the scheme. We do not have that from passive congestion of the intestines. I do not know why we do not. That has always surprised me. We have nothing at all or we have constipation. Poor appetite is the first digestive symptom we have had.

We have to verify the yellowish tint to the skin. There is no one point in histories that is so often not true as "jaundice." When a patient says to me, "I was jaundiced five years ago," I write it down, but I do not believe it. It generally means that he looked a little sallow. But it says "a yellow tint in the sclerae." If that is true it is jaundice.

This pain with the jaundice, if it is jaundice, makes us think of gall-stones. We can perfectly well have some gall-stones and some renal disease. This may not be the thing we are thinking of. Or this may be passive congestion in the liver with the pain in the liver that we get from that congestion.

We have been talking about gall-stones as a possibility. Can gall-stones cause orthopnea? No, they cannot. If he had orthopnea he had something other than gall-stones. So I will revert, until we get further evidence, to the belief that this man is going to turn out to have heart or kidney trouble.

We are not told whether the nycturia is a new or an old symptom. If it is a new symptom it is evidence of passive congestion of the kidney or of nephritis. I have learned from these exercises that nocturnal urination can go with a kidney perfectly normal except for congestion, without prostatic trouble and without any local cause of irritation.

Miss Painter: The nycturia had lasted for the past ten days.

Dr. Cabot: If the record is correct then, as we have no reason to doubt, this is a nocturnal urination of ten days only, not a habit, and therefore evidence of passive congestion of the kidney or of nephritis.

This is one of the gains of weight that we are sorry for. Of course it means edema.

His stopping work gives us an idea how badly off he was at that time.

All the lung signs were on the right side. It sounds like fluid in the right chest, and if we follow up the history, fluid probably dropsical and not inflammatory,—in other words probably a right hydrothorax. Right hydrothorax is commoner than left or bilateral hydrothorax.

Unless the dilated veins are more exactly described we cannot tell whether they are of the type of vein that makes us think of portal obstruction or whether they are not significant. Many people have some dilated veins, but they are not characteristically placed.

### NOTES ON THE PHYSICAL EXAMINATION

He did have icterus.

Presumably there was decreased or absent breathing. If all these other chest signs are there probably the breathing is decreased as well. That backs up the idea that there is fluid in the right chest, and a good deal of fluid, more than we think of in a simple hydrothorax. We begin to think of some local obstruction.

"Increased bronchovesicular breathing" is a bad term. "Increased" does not mean anything, because bronchovesicular breathing is not normal. We should say "loud." That is what we hear when one lung is doing the work of two; one lung is overworking because the other lung is out of commission.

I have no idea what râles "pleuritic in quality" means. The man who heard them thought there was pleurisy, but there is no such thing as a pleuritic râle so far as I know.

In the heart examination we have to ask ourselves, Is this an enlarged heart or a displaced heart? He has fluid in the right chest, and although fluid in the right chest does not displace the heart as much as fluid in the left, it is still displaced by fluid in large amount. So this can be a perfectly normal heart pushed over for all we know yet. The right border of dullness could not be determined of course because of the dullness of the whole right chest. So until we get other evidence we shall not know whether the heart is enlarged or displaced. The record, Miss Painter says, gives us the information that the man who examined the heart thought it was displaced and not enlarged.

This blood pressure means that earlier he had probably had a hypertension. It is on the border-line of hypertension now. 100 is the borderline between a diastolic pressure that is too high and one that is not, and we always pay more attention to the diastolic than to the systolic. So I should say he probably had had a previous high pressure and probably has a little now.

The electrocardiogram means a big left ventricle or a displaced heart, either of which goes along with the high blood pressure and with the hypothesis that we have been entertaining all through.

There is ascites.

I take it that the man who wrote this account thought there was a portal obstruction.

I think we may say essentially an afebrile case.

A normal kidney is what this urine report says. So far as the record goes it is against what I have been thinking of. We have then, so far as this is concerned, unless we get some new evidence later, to say this is not nephritis. It is passive congestion of the kidney perhaps, but perhaps the liver will have to be considered very carefully in view of what they say about the abdomen.

The hemoglobin may be seventy per cent., but I do not believe it. It is a very rare finding. My guess is that the house-officer was hedging. We get it above that and below that, but very rarely seventy per cent. The smear is normal, which means that the hemoglobin is normal. So I believe it was 100%, only they did not like to say so. With a sick patient they do not like to say that the hemoglobin is all right.

The non-protein nitrogen is a little high, but I do not think outside the normal limits. It is above the normal limits that Folin gives, but I think we get such a figure here with normal kidneys many times.

The tests on abdominal tap fluid do not help us. It is perfectly proper to do that examination, but it merely tells us that there is a transudate in the abdomen, not presumably an exudate, and that it comes either from the liver or from the heart.

The chest tap gave a great deal more fluid. It was over three liters, which is a very large chest tap. Again the tests are perfectly natural for a transudate, and that is all we can say. It is very surprising that they got so little fluid from the abdomen with all this dullness. At the second chest tap it had turned into pus. Whether they infected the chest or whether it came naturally I do not undertake to say. But I should think they would have felt uncomfortable when they saw those polynuclears. I do not know much about colon bacilli in a chest fluid. They must have been put there, don't you think, Dr. Wright? It tends to show that that was contamination,—I mean that it was due to the technique.

In the X-ray of course they were looking for cancer.

They are thinking of cirrhosis of the liver and therefore of a big spleen. They did not feel the spleen, which is surprising and makes us wonder whether this is not neoplasm rather than cirrhosis.

We should not have been told about the X-ray treatment of the chest, because if they are giving X-ray treatment it means that they thought he had malignant disease there. I was beginning to think that too, but that is one of the things we should not have been told. Perhaps they are wrong. The treatment was not particularly successful, since the patient died two days later. We are often asked why we do not pay more attention to therapeutics in these exercises. I say that therapeutics so quickly followed by death is not the most interesting kind of therapeutics from the practical point of view.

### DIFFERENTIAL DIAGNOSIS

This is an interesting case because it ends so differently from the way it began. I said, perfectly correctly, that the chances were ninety-nine out of a hundred that it would be a heart or kidney case. But it certainly is not a kidney case, and I do not believe that it is a heart case. There is very little in the examination of the heart to explain the other physical signs. And if he had heart disease why did he die? There is no evidence that he died of passive congestion, no evidence that he died of angina or of embolism or of any of the things that we can connect with the circulatory system. He did not die a cardiac death.

Our attention was early directed to the possibility of obstruction of the portal system by those dilated veins in the abdomen. But we do not know much about the liver. It seems to me that with the jaundice, with the pain he has had there, with the ascites—although it is rather small in amount—and with this recurrence of the large amounts of fluid in the right chest we have to suppose something wrong in the liver.

I have a feeling also that there is something wrong in the right chest.

My impression is that this man died of malignant disease. Why? Chiefly by exclusion. He is at the proper age. He has edema to be explained either by heart, kidney, or local pressure. I cannot explain it by heart or kidney disease, therefore I come to local pressure. The commonest cause of local pressure is malignant disease. I think that malignant disease is in the liver. I am not sure of it. I think it is also in the chest. I am not sure of that. If it is not in the chest why did they get bloody fluid in the tap? If I remember right there was a positive guaiac in that tap. That therefore tends to support the idea that this malignant disease has invaded the right chest as well as the liver. I feel rather surer of the right chest than I do of the liver, but I think it is in both places.

I cannot be any more definite. I do not know whether it started in the right pleura or right lung or in the glands of the chest. Those are the three places where we often see malignant disease started. Perhaps it is not in any of them, though I think it is, and I think it is in the liver.

The heart may perfectly well be a little enlarged. We know it is displaced but it may be enlarged also. I do not think it will show anything else of importance. I do not think the kidneys will show anything of importance. I do not think the left lung will show anything of importance. I do not believe the other organs will show anything of importance unless metastases from the focus of malignant disease which I assume to be present.

Of course there are many other rarer causes of the obstruction which I am assuming is the cause of his dropsy, but I do not think they are important enough to spend time in discussing.

DR. JOHN R. CARTY: In the X-ray plate there is uniform density on the right side of the chest which extends from the apex to the bottom of the lung, obliterating the costophrenic angle,—almost the same density as the rib. The heart and mediastinal contents are displaced to the left. The question which Dr. Cabot brought up, whether or not the left ventricle is hypertrophied, is difficult to answer. If it is hypertrophied at all I doubt whether it is hypertrophied very much. There may be slight hypertrophy.

DR. CABOT: Is there anything from the point of view of technique to show whether that opacity of the chest is due to fluid or solid, or to fluid and solid?

DR. CARTY: We have no way of definitely saying that, although with such a uniform density the chances are that it is fluid. We should say fluid, and possibly some underlying new growth in the lung.

DR. CABOT: There is entire absence of fluid on the left. We see the angle between the diaphragm and the rib. It does not seem as if there was any fluid. If it was a dropsical fluid on the right there would certainly be some down there on the left too. It certainly is not inflammatory fluid. We had the evidence of that on the first tap. It became inflammatory after that. So it seems as if it must have been from new-growth.

DR. CARTY: Dr. Camp thought that the stomach and the first portion of the duodenum were negative.

A PHYSICIAN: Do you think it was primary in the lung?

DR. CABOT: I do not know. Of course primary malignant disease in the liver itself is rare. It seems to me there is more evidence of trouble above the diaphragm than below, and that would lead one to say probably primary below.

A PHYSICIAN: Do you think there was anything below?

DR. CABOT: The fact of jaundice and the pain in the liver made me think there was something in the liver. It is perfectly possible that it is all above.

DR. E. L. YOUNG, JR.: If there was a new growth in the right chest wouldn't that make enough venous obstruction to cause the congestion in the abdomen?

DR. J. H. WRIGHT: I do not see why it should.

DR. CABOT: I can think—can't you?—of very extensive processes in the right chest without anything in the abdomen.

A PHYSICIAN: Was the stomach negative by the chemical tests?

DR. CABOT: We did not go into the chemical tests. I am not sure that they were made. But by the X-ray it was negative. In our experience in France, where we had an extraordinary number of examinations, we found that if we neglected the chemical examinations we got on all right. We wanted to do them, but we had not time. If the X-ray man said the stomach was all right it was.

### X-RAY INTERPRETATION

The findings suggest a large pleural effusion. Possibility of primary lung tumor however should be kept in mind.

### CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Malignancy, possibly lymphosarcoma of chest or carcinoma with metastasis.
Ascites.
Hydrothorax, right.

### DR. RICHARD C. CABOT'S DIAGNOSIS

Malignant disease of the chest and liver.
Hypertrophy of the heart?

### ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

Cirrhosis of the liver.

2. *Secondary or terminal lesions*

Hypertrophy of the spleen.
Icterus.
Dilatation of veins of the abdominal wall.
Hemothorax, right.

3. *Historical landmarks*

Chronic pleuritis.

DR. J. H. WRIGHT: The examination was restricted to a small incision in the abdominal wall, but the nature of the disease was determined. The liver was one-half the usual size. The capsule showed some thickening, was glassy, and rounded up over innumerable smaller and larger nodules, giving a finely to coarsely granular aspect to the surfaces, which were hobnailed in appearance in places. On section the tissue was tough and thickly sown with smaller and larger nodules set in a grayish fibrous meshwork. The nodules were grayish brown, and in many instances were of a dull bluish-green-brown color. No definite adhesions were made out. Microscopically these were the typical appearances of cirrhosis of the liver.

The right pleural cavity contained 2000 c.c. of thin bloody fluid. The left was negative.

The heart was rather small for him. The remarkable feature was the enormous dilatation of the vessels of the anterior abdominal wall between the pubes and the upper border, along a broad strip up and down. The vessels here were of great size, some even admitting the finger.

The spleen was about three times as large as usual.

DR. CABOT: There was nothing at all wrong in the right lung?

DR. WRIGHT: No, so far as we know. The right lung was collapsed, the left lung negative.

DR. CABOT: This case interests me very much, because I happen to remember a similar one in the wards some years ago. We all had a crack at the diagnosis, and we were all wrong except one man, and that was Dr. Wright. We had as in this case a recurrent right-sided hydrothorax, and we tapped and tapped and tapped and always got fluid. And for some reason or other Dr. Wright was the only man who knew that it was cirrhosis of the liver. Why there was such a lot of fluid on the right we did not know in that case and we do not know in this. I thought it was just a freak the first time, but now that we have had two cases somebody ought to work out the connection between cirrhosis of the liver and a large recurrent hydrothorax.

DR. WRIGHT: Don't you think that enlargement of the vessels of the abdomen should have been more stressed in the record?

DR. CABOT: They saw it more in the chest than in the abdomen.

DR. WRIGHT: This was a fat man, and it was apparently an enlargement of the deep ones. Don't you think more importance should have been placed on it?

DR. CABOT: I do, now.

DR. WRIGHT: I do not refer to superficial vessels.

DR. CABOT: It means portal obstruction. There was a mistake in my reasoning in that I did not consider cirrhosis enough. Did you find much ascites? That was one of the things that threw me off.

DR. WRIGHT: There was no ascites. The peritoneal cavity was moist.

DR. CABOT: They got a little on tapping the abdomen, but in the chest they got three liters.

DR. YOUNG: He has an unusually efficient venous anastomosis around the portal circulation. Wouldn't that account for the lack of ascites?

A PHYSICIAN: There is no alcoholic history in this case.

MISS PAINTER: He denied it.

DR. CABOT: We do not account for the absence of ascites. It is not accounted for unless it had come and then gone. But he had, if I remember right, dullness in the abdomen.

DR. WRIGHT: Why could not the great dilatation of the veins have given a collateral circulation sufficient to prevent ascites?

DR. CABOT: That is the suggestion that was made by Dr. Young, and I think it is a good one. There was movable dullness in the flanks and a questionable fluid wave. I certainly thought there was fluid, and there was a little. But I think the suggestion that the big veins took care of the rest is the proper explanation.

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## CASE 11432

### MEDICAL DEPARTMENT

A Scotchman of seventy-three entered the hospital June 2 semi-comatose. The complaint was abdominal pain. The history was given by his wife. His mother and one sister died of "rheumatism," another sister of "shock." As a young man he had several operations on his nose for polyps. Thirty-one and twenty-one years before admission he had hemorrhoidectomy. Twenty years before admission he vomited a pint of blood, but recovered quickly. During the past few years he had had several attacks of tonsillitis and bronchitis. He had had no sore throat for five years. He urinated two to four times at night. For the past two years he had been treated in the Out-Patient Department of this hospital for bladder trouble associated with marked frequency. He passed a little bloody urine. He had some tendency to retention. His bowels were constipated. He was very nervous, occasionally dizzy. For a year or two he had cramps in his legs, feet and hands.

During the past few months he had had sharp generalized abdominal pain, usually at

night, occurring every week or ten days with no apparent relation to food previously taken, and lasting for an hour or two. Four days before admission he became very nauseated after supper and had sharp abdominal pain chiefly on the right side radiating around his waist. He went to bed and felt much better. His wife noticed that he breathed rather slowly during the night. The next morning on returning from the bathroom he almost fell down. He stayed in bed during the day and felt much better. During the night he became nauseated and vomited almost every half hour, a greenish yellow material. The sharp, rather generalized abdominal pain recurred, becoming more marked on the right side, radiating around his waist, and continuing the following day, more marked when he moved in bed. A physician prescribed medicine taken by mouth. The patient frequently vomited after taking a little water. His bowels had not moved for two days. An enema produced no results, and another given the day of admission to the hospital by a district nurse gave unsatisfactory results. Since the onset of the pain he had had rather constant mild headache and had been drowsy.

Records of the Out-Patient Department show that he came in February, eight years before admission, complaining of pains in the bladder region and rectum and frequency. He had suffered from piles for years. A gonococcus test was weakly positive. A Wassermann was negative. A diagnosis of urethral stricture was made. For about ten months the patient made weekly visits to the clinic. The urine was very hazy. He was worked up to sound Number 29. Two years later a right spermatocele was tapped, with the removal of an ounce and a half of material. Two years before entering the hospital he came to the Out-Patient Department about every month. The urine was hazy. Sound Number 30 was passed and nitrate of silver irrigations given. Four years before admission he came to the Nerve Clinic complaining of cramps in the legs and fingers of two or three years' duration. The October before admission definite Romberg was found. A diagnosis of diverticulum of the bladder was made. In February there was tightening at the neck of the bladder. The urine was cloudy. A lumbar puncture gave fluid under normal pressure with normal dynamics, showing three cells, alcohol and ammonium sulphate positive, Wassermann negative. April 2, two months before admission, the urine was very cloudy. There was residual. Sound Number 25 was very tight. A blood Wassermann was negative.

Examination showed an old man with deep asthmatic respiration and ammoniacal breath. The throat and mouth were dry. The apex impulse of the heart was not found. The midclavicular line is not recorded. The percussion borders were 10 cm. from midsternum on the left, 3 cm. on the right, the supracardiac dullness 7 cm. The artery walls were palpable, the temporals and brachials tortuous. The blood pressure was 90/70. The lungs were hyperresonant, with râles throughout. The abdomen showed tenderness in the right flank and was full of fecal masses. Rectal examination showed the prostate slightly enlarged, not hard. The knee-jerks and ankle-jerks were not obtained. Romberg was not attempted.

The temperature was 102.4° to 99°, the pulse 140 to 100, the respiration 43 to 36. The amount of urine is not recorded. The specific gravity was 1.016. A specimen taken in the Emergency Ward was loaded with leucocytes. A later specimen showed 15 to 20 leucocytes per high power field and a slight trace of albumin. The renal function was zero. The hemoglobin is not recorded. The leucocytes were 11,200, the polynuclears 77 per cent., the reds 3,837,000, with normal variation in size and shape and slight central achromia. The platelets were slightly reduced. An analysis of the blood fasting showed the nonprotein nitrogen 68 mgm., uric acid 6.0 mgm., creatinin 3.3 mgm.

Orders: June 2, soapsuds enema, soft solid diet, force fluids, magnosium sulphate ℥ i daily in the morning, two compound cathartic pills with half an ounce of brandy at once. June 3 adrenalin minims v s.c., digifolin gr. ii (two ampules) s.c. at one, five and nine p. m., croton oil one drop intramuscularly, morphia gr. 1/6, repeat at the end of one hour if necessary. June 4 digifolin gr. iii t. i. d., morphia gr. 1/8 s.c.

The patient was given fluids intravenously, as he could retain nothing by mouth. The day after admission his heart began to fibrillate, with rapid, weak, irregular action. The patient did not rally, went into deepening coma, and June 4 died.

## Discussion

### by dr. richard c. cabot

### notes on the history

The vomiting of a pint of blood would be important if we were sure of it. As we were not there to see it I do not feel sure that it was as much as that.

The slow breathing was probably Cheyne-Stokes.

His weekly visits to the clinic were presumably to stretch the stricture. Is the Number 30 sound a pretty good size?

Dr. Young: Yes, that is a good size.

Dr. Cabot: Is there any connection between the spermatocele and the other?

Dr. Young: No.

Dr. Cabot: The silver nitrate irrigations were presumably for a cystitis?

Dr. Young: Yes.

DR. CABOT: Of course a Romberg at seventy-three means a great deal less than it does with other people. Old men frequently cannot stand without swaying because their legs are weak. Of course with this pain we are thinking of the gastric crisis of tabes and a Romberg from that.

They must have cystoscoped him.

This is a negative spinal fluid on the whole, I should say.

Before he is examined, what are our guesses? He has had a chronic trouble in his bladder, urinary sepsis there, and I should suppose a good deal of back pressure on his kidneys. I should suppose that his kidneys are a good deal damaged from that, and also very likely infected.

DR. YOUNG: They are almost certain to be infected with such a showing.

DR. CABOT: Can they account for everything in this case? I cannot connect the pain, though of course he might have pyonephrosis. But I am not accustomed to see this kind of pain. So that the pain still remains not easily explained. The mild headache and the drowsiness may perfectly well come with uremia, which would explain his vomiting, the whole depending on his urinary sepsis and damaged kidneys.

### NOTES ON THE PHYSICAL EXAMINATION

All of this examination goes to back up uremia.

The percussion measurements are normal. Is anything given about heart sounds or murmurs?

MISS PAINTER: There were no murmurs; the sounds and action were normal.

DR. CABOT: We have a negative heart, then, so far as the examination goes.

I don't think we can get much out of his blood pressure. He is near death.

Perhaps his abdominal pain was due to chronic constipation and the colics that would come with that. On the other hand, of course, we cannot help thinking of intestinal obstruction.

If he has a stricture of the urethra, and we have good evidence that he did have, he will not have a big prostate, and so we will say that he has not got a big prostate even though somebody thought he felt it.

Absent knee-jerks and ankle-jerks of course make us think of tabes, as do the Romberg and the abdominal pain. Nothing is said about his pupils, but failure of reflexes cannot be taken very seriously in a man semiconscious and near death, and I do not so take it. If they were thinking of tabes his cramps might be lightning pains.

The temperature probably went with the infection of the urinary tract that we have assumed.

A renal function of zero ought to mean something. We do not take a great deal of stock in lowered renal functions. Five to ten per cent. even can go along with normal kidneys. But when they say zero we think there is something wrong, especially when they say no edema, as in this case. Ordinarily we do the renal test here subcutaneously. So there is always a possibility that the dye is not absorbed and so does not come through. But in the absence of heart trouble I think it was absorbed, so "zero excretion" backs up what we have been thinking of as the real cause of his trouble.

The red count goes very well with chronic nephritis.

The non-protein nitrogen is rather high, but not so high as we should think.

The uric acid and creatinin are both high.

This is the treatment of a uremic patient.

### DIFFERENTIAL DIAGNOSIS

The chief thing that worries me is his abdominal pain. The rest of the case goes pretty well for a diagnosis of urinary sepsis, chronic nephritis, uremia and death. But I do not know how to account for his abdominal pain. It may be all due to constipation. That is on the whole the best I can say. I am a little worried for fear it may have been intestinal obstruction.

DR. G. C. CANER: Could that pain come from the heart?

DR. CABOT: Of course we might have an infarction of the heart muscle, but his heart seemed to be in pretty good shape until the end. We did not get the pulse failure that ordinarily goes with infarction.

DR. CANER: He has a very small pulse pressure.

DR. CABOT: Ordinarily too infarction pain is in the middle rather than on the right. This is described all on the right side. It is a good thing with an old man with pain in that region always to think of cardiac infarction. But it seems to me we have rather little to support it here. I am unable satisfactorily to account for the abdominal pain.

A PHYSICIAN: Could sclerosis of the abdominal vessels have started a spasm in them?

DR. CABOT: Nobody can deny it because nobody can be there to see. It is a hypothesis we use in such cases. I have always thought we should be able to work out a better clinical picture of abdominal arteriosclerosis than we have. We know something of what it does in the brain, heart, arms and legs, and the kidneys. But so far I do not think there is any clear picture of abdominal arteriosclerosis. With spasm in the vessels of the abdomen as you suggest, I should say very possibly we might have such pain. I do not know anything about it.

A PHYSICIAN: Would you say renal colic?

DR. CABOT: It is not in the characteristic place. It does not seem as if this pain was

typical of that. I tried to explain it in the beginning as the pain of pyonephrosis or hydronephrosis, but it does not seem to go with that. Of course in eclampsia, which is a first cousin of uremia, we often get abdominal pain. I do not know how it is accounted for, but I have not seen such pain in uremia.

I think he has chronic urinary infection with back pressure in the kidneys resulting in a sclerosed, non-functioning set of kidneys with or without pyonephrosis or hydronephrosis. He probably has a hypertrophied and dilated heart with arteriosclerosis. I do not think that plays any part in his death. The cause of his abdominal pain I do not know, unless it is due to constipation.

A PHYSICIAN: Could those cramps come from uremia?

DR. CABOT: I think they could come from arteriosclerosis, which we think he has. But they could also come from uremia. I think arteriosclerosis is a symptom. If we explain the peripheral cramps from arteriosclerosis we might perfectly well adopt the suggestion of saying the same thing in the abdomen,—a vascular spasm.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Arteriosclerosis?
Pyonephrosis.
Uremia.
Bronchopneumonia.

DR. RICHARD C. CABOT'S DIAGNOSIS

Urinary sepsis.
Chronic nephritis.
Hypertrophy and dilatation of the heart?
Arteriosclerosis.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

Purulent cholecystitis with areas of necrosis in the bladder wall, purulent pericholecystitis and purulent peritonitis in the region, and fibrinous peritonitis elsewhere.

2. *Secondary or terminal lesions*

Diphtheritic esophagitis and duodenitis.
Edema of the lungs, with hypostatic pneumonia.
Slight arteriosclerosis.
Cholelithiasis.
Soft hyperplastic spleen.

3. *Historical landmarks*

Chronic pleuritis.
Focus of obsolete tuberculosis of the left lung.
Slight hydronephrosis, lower pole of the left kidney.

DR. J. H. WRIGHT: The peritoneum in the region along the course of the intestines showed injected patches and fibrinous material. In the region of the gall-bladder the peritoneum was red and coated with exudate which extended up over the liver. The appendix was negative. The gall-bladder was much enlarged, twelve centimeters long by thirteen in circumference. The mucosa showed numerous smaller and larger patches of purulent necrosis. The wall was thickened. Beneath the patches the condition extended through to the peritoneal surface of the bladder. In the peritoneal surface of the bladder there were discrete and confluent patches of purulent material. The bladder contained two small stones, and there were two in the cystic duct. The cystic duct otherwise was negative. The hepatic and common ducts were slightly dilated. Their mucosa was negative.

The pancreas and duct of Wirsung were negative.

The kidneys were of fair size. The capsules stripped. The only lesion made out was in the region of the lower lobe, where the surface was irregular, with some dilatation of the calices. The condition of the kidney is summed up as slight hydronephrosis in the lower pole. In the bladder there were some small diverticula.

DR. CABOT: Was the other kidney all right?

DR. WRIGHT: Yes. As a secondary inflammatory phenomenon there was diphtheritic esophagitis and duodenitis. That was the cause of his abdominal pain.

DR. CABOT: The hemorrhagic hepatitis of eclampsia is inside the liver rather than stretching the capsule, isn't it?

DR. WRIGHT: The lesions are very spectacular in the liver.

DR. CABOT: Ordinarily we do not explain anything as causing pain in the liver unless stretching the capsule. But if these hemorrhages do come under the capsule and all through the liver, I think that might account for it.

DR. YOUNG: Were the diverticula very deep in the bladder?

DR. WRIGHT: No, they were very small.

DR. CABOT: Note the zero renal function without nephritis!

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## CASE 11433

SURGICAL DEPARTMENT

A married woman sixty-three years old entered May 8 complaining of vomiting. A daughter-in-law and a granddaughter had tuberculosis. The patient's past history was essentially negative except that she had had a tired feeling in her head for years, used two pillows for sleeping, and had slight swelling of the right ankle.

For nearly a year she had been feeling tired

and losing weight and some strength. During the past winter her bowels had been constipated. April 2, a month before admission, she had an attack of epigastric cramps lasting two or three hours and making her stop work. Two hours after the pain ceased she vomited twice. During the next week she felt fairly well. April 9 she began to have steady rather projectile vomiting of considerable brown fluid even though she did not eat. At first she vomited two or three times in twenty-four hours, more in the evening than in the morning. There was no nausea or pain. For four weeks her urine had been brown and the stream somewhat difficult to start. Two weeks before admission she went to a hospital, where she had less vomiting. She believed that her temperature had been subnormal and that X-rays showed "shadiness of the gall-bladder" but nothing wrong with the stomach, heart or lungs. Jaundice was first noticed two weeks before admission. Over a year before admission she weighed 165 pounds, her best weight. Her present weight was 131.

Examination showed a very jaundiced, poorly nourished, sick looking woman. The skin was extremely dry, scaly, thickened and inelastic. The sclerae were yellow. The teeth were very poor. There was pyorrhea. The abdomen was lax and doughy. On deep inspiration a mass could be felt in the region of the gall-bladder. There was no tenderness. Pelvic examination showed slight reddening around the urethra. The uterus was indefinitely felt, slightly retroverted. The vaults were slightly tender. There was a questionable small mass on the left. Rectal examination showed internal hemorrhoids.

At entrance the temperature was 100°, afterwards 97.5° to 99.1° until operation. Before operation the pulse was 68 to 100, the respirations normal. The urine showed the slightest possible trace to a trace of albumin at two of three examinations, specific gravity 1.010 to 1.020, amount normal, bile at two examinations, leucocytes at two. The hemoglobin was 70 per cent., the leucocytes 12.000, the polynuclears 76 per cent. The bleeding time was 3 minutes to 3½ minutes. The clotting time May 12 began in twenty-eight minutes, complete in forty minutes, May 14 calcium tube thirty-six minutes, began in forty minutes, complete in an hour and twenty minutes. The non-protein nitrogen was 36 mgm. NaCl in plasma 474 to 432 mgm. per 100 c.c., calcium 9.7 mgm. to 9.5 mgm. There was 350 c.c. of vomitus, dark brownish fluid with a gastric odor and containing a large sediment of coffee ground material. The filtrate was a clear watery greenish fluid. The precipitate was dark reddish-brown flocculent material giving a very strong guaiac. On the unfiltered vomitus the iodin test for bile gave a very faint green ring, on the filtered no ring. X-ray films of the gall-bladder made at fifteen and eighteen hours after the Graham test failed to show any shadow which suggested the outline of the gall-bladder. The dye apparently had not entered it. The stomach was low, atonic and sluggish. No irregular or tender points were noted. There was a small amount of retention from the six-hour meal. The first portion of the duodenum filled readily. There was a filling defect involving the second or descending portion of the duodenum. The third portion of the duodenum was fairly well seen. The motor meal was in the transverse colon. There was no definite evidence of organic disease of the stomach or the first portion of the duodenum.

During her first three days in the hospital the patient vomited only once. May 14 after having 10 c.c. of ten per cent. calcium chlorid intravenously she immediately felt very warm and vomited considerably.

May 16 operation was done. She was very uncomfortable for the next two days, vomiting black coffee ground material showing a very strongly positive guaiac. The stomach was washed by the use of a nasoduodenal catheter and 28 ounces obtained. May 20 she seemed a little better and had not vomited so much. The vomitus was not black. May 21 she died.

## Discussion

BY DR. EDWARD L. YOUNG, JR.

The past history does not tell us very much, because vomiting can be the symptom of so many different things, from something acute to something of longer standing. It does not carry us far. And tuberculosis is more interesting when our knowledge of the intimacy of the patient with it is known rather than the relationship of the people having it. So that does not help us much.

"Projectile vomiting" suggests that there is either reversed peristalsis or some pyloric obstruction, but it does not tell us anytihng very definite. Of course we often associate it with brain tumor, but I think it is a poor term.

On the chances this is a concentrated urine, which is always an acid, burning urine, and it may mean also that there is bile in it.

There is a loss of thirty-four pounds within the last year.

So far this is definitely pointing toward the upper abdomen, and with the jaundice a suggestion of trouble in the biliary tract.

When these masses come into sight in these examinations we always wonder whether it is a mass that is felt because the history suggests that there may be trouble, or whether the thing is definitely felt. Where as here it suggests that it is somewhat indefinite I think we always have to be more sure of it before we put full faith in it.

One very interesting thing about diseases of the biliary tract is the various ways in which

symptoms can come. For instance, in the past year I have seen three or four cases in which the patient was supposed to have had heart trouble for a number of years in spite of the fact that no definite organic lesion of the heart had been demonstrated. One patient for example had palpitation, dyspnea and fainting spells at night, and nothing had ever been proved so far as the heart was concerned, and it was not until one attack of gall-stone colic led to the study of that region that they were found. So far all of the patients have been cured of their heart attacks by operation. Symptoms of that kind are a pretty long cry from symptoms pointing to the biliary tract. Of course we know that a story such as this of vomiting, of loss of weight, even without the more direct evidence of jaundice, can come from trouble in the gall-bladder and in the liver.

As we see this woman we have evidence that is pointing toward the biliary tract, and according to the X-ray there is no trouble in the stomach or duodenum. So that if we put the whole picture together it seems as if we had to make a diagnosis of cholecystitis with or without stones, and of course the accompanying hepatitis. That would fill the picture here, and I think it would account for all the symptoms which she has been having for the past year.

The chemistry does not show anything badly out of the way. The only thing we are a little afraid of is the slowness in clotting time.

The treatment of course is the removal of trouble. We do not know whether there are any stones here or not. It is not necessary to have stones in order to have this amount of trouble. An old cholecystitis which has contracted with thickening of the gall-bladder, with obstruction, with the infection in the liver which goes with it, is all that is necessary.

The X-ray simply shows that it is an abnormal gall-bladder in that there is no shadow shown.

It seems to me the only thing to do is operation, and here they felt that the preliminary use of calcium chlorid was necessary. In the Mayo Clinic they use that as a routine for two or three days before operating. Here it was apparently given only once. There are certain cases in which there is a slight reaction from calcium chlorid, but other than that no harm that we have ever met with here coming from the use of it.

I do not see that we are justified in making any diagnosis other than cholecystitis, and of course with the finding of that the best treatment is the removal of the gall-bladder. This has been going on a long time, and there comes up the question whether there should be drainage to include drainage of the liver. She has jaundice, and it may be that there is a stone in the common duct and that the common duct will be dilated, and that drainage from the common duct will be done. It may be that the process is a starting process, and in that case drainage from the stump of the cystic duct may be worth while, or no drainage at all.

Of course we cannot say that this is not a carcinoma. It has been going on only a year definitely. She has been losing weight and strength, and although that loss of weight can be easily accounted for by the upset of digestion, it may be that back of it there is malignancy of the gall-bladder itself or of the duct or the papilla, possibly an obstruction due to carcinoma of the head of the pancreas. Nothing is said here about the stools, so I assume that there was not complete obstruction and bile was coming through into the intestine. If it was true that the gall-bladder was felt it would be in favor of an obstruction from carcinoma. So I think that the only thing to do is to operate. If it is a case curable by operation well and good. And if it is malignant disease presumably nothing that can be done in the line of operation will give any chance for cure.

A Surgeon: How do you account for the blood in the vomitus?

Dr. Young: I do not see how it is possible to account for the blood in the stomach on that diagnosis. I do not know whether repeated vomiting can result in congestion enough to have a small amount of blood in the stomach or not. Blood streaks in the vomitus are easily accounted for by the irritation of the esophagus or the pharyngeal wall. On the other hand it is not possible to account for that by carcinoma of the pancreas or by any other condition outside the stomach, any more than it would be by cholecystitis. So I do not think it helps us.

The question here is between a carcinomatous condition and an inflammatory condition, with or without gall-stones. I do not know how to make a distinction.

A Surgeon: Was the urine negative for sugar?

Miss Painter: It was.

### Dr. Young's Pre-operative Diagnosis

(1) Cholecystitis.
(2) Carcinoma of the head of the pancreas.

### X-ray Interpretation

The filling defect in the second portion of the duodenum is somewhat suggestive of a pancreatic neoplasm.

### Pre-operative Diagnosis

Carcinoma of pancreas.

### Operation

Gas-oxygen. Incision to the right of and above the umbilicus. In the body of the pancreas just at the junction of the head there was a hard mass half an inch in length to an inch

in width. It was rather difficult to see how this growth shut off the common duct so completely. The gall-bladder was moderately thickened and slightly edematous. It was dilated, with the escape of very thick black bile, almost the consistency of wheel grease. Apparently the whole biliary system was filled with this material. A cholecystduodenostomy was done with two rows of sutures without a tube. This operation was done in spite of a clotting time of forty minutes. The problem had been discussed with the family and the family urged operation.

### Further Discussion

A Surgeon: Were the stools clay-colored?

Dr. Young: I assume they were not, because there was no mention of it. In other words the operation was just an attempt to side-track bile into the intestine.

Miss Painter: The stools were light brown and greenish.

Dr. Young: Apparently very little bile coming through.

We have a lesion in the duodenum, so that it is easy to account for the blood.

On the chances she died of peritonitis, though it is not necessary to say that.

The X-ray people apparently had some definite feelings about it and said it was suggestive of pancreatic neoplasm.

Dr. John R. Carty: I did not do the fluoroscopic part of this examination, but I think that I can interpret fairly well what the examiner saw. There is a normal looking stomach, peristalsis appears normal, the cap looks smooth. There is apparently no filling defect in the stomach or in the cap. But in the second portion of the duodenum there is a vacant space. As we look at the film it is difficult to tell whether or not there is a defect here, but the examiner watched and evidently there was some contraction or abnormality in that spot. The third portion of the duodenum apparently was all right. Evidently the examiner by manipulation and observation felt that the lesion was extrinsic to the duodenum rather than intrinsic. There is no mention made in these notes of a mass being palpated, but apparently he felt reasonably certain that there was an extrinsic lesion involving the second portion of the duodenum, and he believed that it was probably a pancreatic neoplasm.

Dr. Young: When you have a distortion of the duodenum from the gall-bladder, where does that come?

Dr. Carty: In the first portion of the duodenum. But when we come to distortions of the second and third portions we have to be very cautious, because variation is quite marked in that region. But apparently these findings were quite definite or the examiner would not have committed himself.

The Graham test was done. In fifteen hours after they gave the diet there was no dye in the gall-bladder.

Dr. Young: That spells nothing more than some abnormal condition, does it?

Dr. Carty: It might say that a normal gall-bladder was distended by bile.

### Clinical Diagnosis (from hospital record)

Carcinoma of the head of the pancreas.
Post-operative vomiting.
Cholemia.
Cholecystduodenostomy.

### Dr. Edward L. Young's Diagnosis

Carcinoma of the head of the pancreas.
Peritonitis?
Shock?

### Anatomical Diagnosis

1. *Primary fatal lesions*

Carcinoma of the head of the pancreas.
Chronic pancreatitis.

2. *Secondary or terminal lesions*

Icterus.
Cholecystduodenostomy.
Hemorrhage into the gastro-intestinal tract.
Edema of the lungs.

Dr. Wright: The examination showed a neoplasm of small size in the head of the pancreas, extending to a limited extent into the wall of the duodenum with ulceration, and also involving the lower portion of the common duct with obstruction and dilatation of the hepatic and common ducts above. There was extensive hemorrhage into the gastro-intestinal tract, clotted blood being adherent to the ulcer in the duodenum.

Metastases were inconspicuous. In the region of the head of the pancreas the glands were slightly enlarged and rather firm.

Dr. Young: Was the suture of the anastomosis intact? There was no peritonitis?

Dr. Wright: The anastomosis was in good condition. There was obstruction of the duct of Wirsung producing fibrosis of the pancreas, which is common in carcinoma of the head of this organ.

Dr. Young: The extension of the lesion gives us a cause for the blood in the vomitus the first time.

## INSANITY AND DISABILITY CLAIMS

Insanity was responsible for twenty-four per cent of the approved total and permanent disability claims allowed Prudential policyholders during the last five years, according to an analysis of claims for disability benefits. It ranked only second to tuberculosis, which was the cause of total disability in thirty-five per cent of the cases.—*Prudential Service Bureau.*

THE BOSTON

# Medical and Surgical Journal

*Established in 1828*

Published by The Massachusetts Medical Society under the jurisdiction of the following-named committee:

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## BE NOT THE FIRST BY WHOM THE NEW IS TRIED

MUCH credit is due the commercial houses for their enterprise in developing new pharmaceutical preparations and in devising new apparatus for diagnostic and therapeutic purposes. A few of their productions are real contributions to medicine; many are improvements upon their predecessors. Research work of solid value is being carried on in many commercial laboratories, and without the practical assistance of chemists and engineers so employed, many of the brilliant discoveries of medical men could never be utilized.

This situation, however, is not without its dangers. Too great an activity in the development of new drugs and apparatus may be productive of harm. An energetic research department busies itself in putting forth new products; to justify the expense of its laboratory, as well as to advertise its progressiveness, the company must distribute the new preparations far and wide. Thousands of letters describing new drugs are mailed every day; ten or fifteen preparations of similar properties and of very nearly equal potency are carried in stock and distributed as samples, where one standardized preparation would suffice. As a result of this multiplication of remedies, all adapted to the same end, confusion is created in the minds of the public, the practitioner and the nurse. Instead of becoming thoroughly acquainted with the virtues and limitations of one particular drug, many physicians keep turning to the latest newcomer, only to find it no more potent than those that preceded it. Such, for example, is the situation in regard to the barbital compounds. Another bad result of this policy is the excessive cost of all drugs. The expense of maintaining a large research staff, of heavy advertising of new products, and of carrying many similar drugs in stock, has to be borne by the business as a whole. As usual, the consumer pays.

So far as the employment of new remedies is concerned, it is unlikely that much actual damage to the quality of the practice of medicine results. With new apparatus, the situation is different. A doctor, carried away by the enthusiastic representations of the salesmen, purchases an expensive therapeutic machine, let us say of the electrical type. Having invested in it, he feels that he must use it frequently in order to get his money's worth, so he employs it on many cases in which its use is really not indicated. A typical example of this sort of thing came to our attention recently. A man afflicted with urethritis had consulted a doctor who apparently had fallen victim to all the recent innovations in therapy. For injection the patient had been given a colorless form of argyrol and a new preparation of mercurochrome carried in some sort of paste; further treatment consisted of application of diathermy to the urethra. This treatment had been carried on for five weeks with symptoms of the infection still persisting. Upon examination by another physician, it was found that the posterior urethra was infected, the infection being kept up by a very definite vesiculitis. The disease would never have been cured by the line of treatment which had been given. The point of the story is this: whereas the measures employed by the patient's doctor were theoretically adapted to cure urethritis, they had been applied regardless of the pathology of the particular case. No doubt the doctor felt that he was administering the very latest and (therefore) the very best treatment; but no treatment, no matter how well advertised and sponsored, can succeed without careful preliminary diagnosis and an understanding of the pathology of the disease.

To prevent such occurrences as this, caution should be employed both by the firm putting out the product, and by the physician who listens to the arguments of the salesman. Unless such caution is observed, undue promotion of new offerings will react against both the producer and the physician, and will be a hindrance rather than a help to the patient.

## TUBERCULOSIS IN CHILDREN

THE New York Tuberculosis Association has recently distributed in reprint form two interesting statistical studies from the American Review of Tuberculosis. Godias J. Drolet has reviewed the mortality among children from tuberculosis during the past quarter century in New York, 28,761 deaths from various forms of the disease having been reported among children under fifteen years of age in the last 26 years. The meningeal form causes half of the deaths, the death-rate from this cause, however, having been reduced from 78 to 17 per 100,000, or by 78 per cent. Pulmonary tuberculosis is the next in importance, causing nearly one-third of the tuberculosis deaths, and the death rate from peritonitis, formerly 7 to 8 per 100,000, has, in the last couple of years, dropped to 1. Pasteurization of milk and examination of food handlers has probably been largely responsible for this decline. In total the death rate from the various forms of tuberculosis in infants under one year of age has declined from 609 in 1898 to 94 in 1923, or 85 per cent.

Tuberculous meningitis in children has been made the subject of a statistical study by Beatrice Slayton Herber and M. Alice Asserson. These authors studied 135 cases of the disease in children under 15 years of age, visiting the homes of the families, relatives and neighbors, and investigating 210 tenement houses. The death records of 435 cases in children throughout the Borough of Manhattan were also studied. In only 13 of 135 cases was there no suspicious history of contact, and these authors conclude that the great majority of cases are human in origin, poor housing conditions having considerable to do with susceptibility to the disease. Otitis media, measles, pneumonia and whooping cough seemed also to constitute important predisposing factors.

In general, the great decline in tuberculosis mortality among children must be considered as distinctly encouraging. Improved hygiene and anti-tuberculosis measures must be largely responsible, even taking into account the resistance that may have been acquired by general tuberculization. Despite the low incidence of the bovine type in certain forms of the disease the increased safeguarding of our milk supply must continue.

## THE VALUE OF THE SURGICAL OUT-PATIENT DEPARTMENT IN THE TEACHING OF SURGERY

ANYONE watching the long stream of patients heading for the Surgical Out-Patient Department of one of our large hospitals could not fail to be impressed with the numbers seeking surgical aid as well as the probable wide variety of their complaints. No more fascinating and instructive hospital department is open to the student for his first intimate contact with a widely diversified field of medicine. Here he first learns to do things with his own hands; the actual contact with and ministration to patients under the eyes of a careful instructor give him a foundation for his future work which he never forgets. The practical art of surgery can always best be acquired by the most intimate contact and familiarity with the routine of a well organized clinic. The very multiplicity of conditions encountered give an added charm to the work and incentive to acquire knowledge, both practical and theoretical, which will in his turn enable the student to help humanity through some branch of medicine. The most valuable lesson of all the student learns is perhaps from his observation of the interest and enthusiastic service of his superiors in their chosen work, and of absorbing insensibly something of the atmosphere of work and service for its own sake.

## THE PHYSICIANS' HOME

ATTENTION is called to the announcement of Dr. Robert T. Morris setting forth the plans for "The Physicians' Home, Inc."*

The Board of Directors is made up of men whose names carry the promise of able and discreet management of a worthy project. Here in Massachusetts we have an association which has been aiding those worthy physicians who have fallen by the wayside financially or physically. This organization has performed its function so unostentatiously that its existence has not been generally known. Its resources have not been sufficient to enable it to do more than provide limited service to the needy.

The plan outlined by Dr. Morris will provide more ample assistance for physicians and their families for whom the profession should feel responsible to a large degree. The project has received the official sanction and backing of the American Medical Association and the New York State and County Societies.

Ordinarily we might feel inclined to suggest that since this scheme is designed to be nation-wide it would be wise to have on the board of directors representatives of different sections of the country. This suggestion may not apply at the present time, for the men named have a national reputation and are too big minded to be narrow or sectional in dealing with the problems involved but as time goes on it might be that more interest and coöperation would result from a more diversified representation on this board.

Some person or body must inaugurate the movement and we can safely follow the lead of those who are responsible for the initiation of the plan, for their wisdom will undoubtedly be shown in all necesary details.

We hope that the people of Massachusetts will respond generously.

*See page 800 of this issue.

## THIS WEEK'S ISSUE

Contains articles by the following named authors:

Boos, William F., A.B.; Ph.D.; M.D., Harvard, 1901. Consulting expert in Chemistry, Medicine and Toxicology for various states and the Federal Government. Member of the Industrial Poisons Committee of the National Safety Council. His subject is "My Experience with Certain Industrial Poisons."

Richards, Lyman G., A.B.; M.D., Harvard, 1919. Surgeon in Otolaryngology, Children's Hospital, Boston, and Associate in Otolaryngology, Peter Bent Brigham Hospital. His subject is "A Year of Endoscopy."

Goldberg, Bernard I., B.S.; M.D., Harvard, 1922. Assistant Visiting Physician and Physician to O. P. D., Heart Clinic, Homeopathic Hospital. Assistant in Cardiology, Boston University School of Medicine. His subject is "Value of Specific Treatment in Cardiovascular Syphilis."

White, Leon E., A.B.; M.D., Dartmouth, 1893. Fellow in Otology, Harvard Post-Graduate School. Aural Surgeon, Massachusetts Eye & Ear Infirmary. Member of New England and National Otolaryngological Societies. His subject is "The Optic Canal in Optic Atrophy."

Cronin, Herbert J., M.D., Harvard, 1911. Member Massachusetts Medical Society and American Medical Association. His subject is "Paranoia. A Traumatic Industrial Accident Case Complicated by a Mental Derangement."

Hodgson, John S., M.D., Harvard, 1917. Assistant Surgeon, O.P.D., Massachusetts General Hospital. His subject is "Progress In Surgery."

## The Massachusetts Medical Society

### MEMBERSHIP CHANGES

Dr. Max Bennett of Chelsea has discontinued his office in that city and practises only at 484 Commonwealth Avenue, Boston.

Dr. Theodore C. Erb has moved from 159 St. Botolph Street to 394 Marlborough Street, Boston.

Dr. William L. Kelleher has moved from Worcester (Worcester) to Marlborough (Middlesex South). His office is at 196 Main Street.

Dr. Florence E. H. Knowlton has moved from Sparta, Wisconsin, to New York City, where her address is 121 East Sixtieth Street.

Dr. Samuel R. Meaker has removed his office to the Lister Building, 475 Commonwealth Avenue (at Kenmore Station).

Dr. Guy C. Randall has moved from Hathorne to Salem, 297 Essex Street.

Dr. Ella A. Wilder has moved from Boston (Suffolk) to Middletown, Conn. (Non-Resident List). Her address is 138 Broad Street.

## LEGISLATIVE NOTES

The Joint Committee on Legislation of the Massachusetts Medical Society and Massachusetts Homeopathic Medical Society have unanimously adopted the following statement of their position on matters dealing with the Registration of Physicians in the Commonwealth. It is the intention of the Committee to give this matter the broadest possible publicity. Every Massachusetts reader is urged to study this statement and bring the facts before the public, in order that they may clearly understand the purposes of the Medical Societies.

### Medical Registration in Massachusetts

Prior to 1894 the Commonwealth did not attempt to regulate the right to practice medicine. At that time the Board of Registration in Medicine was established. It provided first that those who had been in practice within the State could continue in practice; second that those who were just graduating from medical schools could be licensed without examination; and third that in the future those who wished to practice medicine must be examined by a Board of seven physicians, not more than three of whom should belong to any one chartered medical society. The Governors for a number of years interpreted this provision to mean that there should be three members of the Massachusetts Medical Society, three of the Massachusetts Homeopathic Medical Society and one member of the Eclectic Medical Society. At a time when the Eclectic Society had become very small and the Osteopaths were moving for a separate Board of Examiners, an Osteopath was substituted on the Board for a member of the Eclectic Society. This situation has continued up to the present day.

The Supreme Judicial Court has decided that the practice of medicine means essentially the treatment of the sick or injured, regardless of any method under which the treatment is rendered. Thus Massachusetts ever since the establishment of the Board of Registration has maintained a single standard of examination for those who wish to practice the healing art. In this respect, the standard in Massachusetts has been high and we have not been exposed to such scandals as occurred recently in Connecticut where men had been licensed to practice medicine by some of the special Boards of Examiners.

During the summer and autumn of 1924 a Recess Committee of the Legislature held hearings and presented a report upon matters having to do with registration including the registration of physicians. The essence of their recommendations so far as it concerned physicians was as follows:

1st. That the single standard of examination

should be maintained and that therefore there should be no special Board of Chiropractic examiners.

2nd. That a Board of seven members should be appointed, no two of whom should belong to any single trade or profession, to have general supervision of the different technical examining boards which had to do with those registered in various trades and professions.

3rd. That this board should be given the power to pass upon the qualifications of medical schools.

4th. That the restriction placed upon appointment of membership in the Board of Registration in Medicine mentioned above should be removed.

The Joint Committee on Legislation of the Massachusetts Medical Society and Massachusetts Homeopathic Medical Society endorsed these recommendations of the Recess Committee and favored the bills accompanying the report until, in the final drafts, they were changed radically. When the power to pass upon the qualifications of medical schools was removed and when a change was made which compelled the Governor to appoint one osteopath on the Board of Registration the bills were opposed by the Joint Committee.

We still favor the recommendations made by the Recess Committee except the second which provides for the creation of a board to be placed over the Board of Registration. We ask that the single standard of examination for the right to practice medicine be maintained. We oppose all efforts to have special Boards of Examiners appointed because we believe that the creation of special boards would inevitably lead to such scandals as have occurred recently in Connecticut, and at times in various States.

We believe it is essential that those who undertake to treat the sick should know the structure of the human body and the manner in which the various organs perform their function. In other words that a knowledge of anatomy and physiology is essential. We regard as essential an understanding of the changes which occur in the various organs when the human machine is out of order or in other words a knowledge of pathology. We consider as essential a knowledge of the methods by which the human body becomes disordered. Thus a knowledge of bacteriology is necessary. We consider it essential that those who are to undertake the practice of Medicine should know the elements of Surgery, or in other words understand those disorders and injuries which require mechanical methods of treatment for their repair. We believe all physicians should understand child birth. These are the important matters. It is important to note that the subjects in which candidates are examined have been determined by the Legislature and not by the members of the Board of Registration.

We consider that the work of our National, State and municipal health authorities which are created to maintain the public health and to prevent disease depends upon the fitness of individual physicians.

We believe that the orderly and efficient conduct of life, accident and health insurance, the work of our Medical examiners in detecting and preventing crime, the work of our school physicians in preventing epidemics, are all dependent upon the fitness of the individual physician. We do not believe that the State should dictate the methods by which the sick are to be treated except in so far as to insist that the public health be not endangered, but we do believe that the State should certify to the competence of all who undertake the treatment of the sick or injured.

We strongly favor the recommendation of the Recess Committee that the equipment of the medical schools and the quality of their instruction be passed upon by some authoritative body. We are ready to have this done by the Board of Registration in Medicine, by the State Department of Education or by any Board of Laymen or Physicians appointed by the Governor. We ask this because at the present time there are only five of the lowest grade medical schools in the United States. Two of these are now under indictment for fraud and may very likely pass out of existence. Two of those which are regarded by physicians as belonging to the lowest class are located in Massachusetts. Graduates of the Massachusetts College of Osteopathy are also examined for the right to practice in this State. The plea is made in behalf of these schools that they give an opportunity for the poor boy to study medicine which would otherwise be denied him. We do not ask or seek the destruction of these schools. We ask simply that they be placed under some supervision by the Board of Education or some other similar body which may require reasonable standards of fitness in equipment and quality of instruction. We do not seek state control of education. The reason why we believe that these schools should be placed under competent supervision is shown by the following statistics, taken from the official reports of the State.

TOTAL NUMBER OF PHYSICIANS EXAMINED, REGISTERED AND REJECTED BY THE BOARD OF REGISTRATION IN MEDICINE FROM 1911-1924

| | Examined | Registered | Rejected | Per Cent. Rejected |
|---|---|---|---|---|
| Harvard | 700 | 692 | 8 | 1.14 |
| Tufts | 969 | 895 | 74 | 7.5 |
| Boston University | 207 | 197 | 10 | 4.83 |

| | | | | |
|---|---|---|---|---|
| Massachusetts College of Osteopathy | 303 | 129 | 174 | 57.4 |
| College of Physicians and Surgeons, Boston | 156 | 55 | 101 | 64.7 |
| Middlesex College of Medicine and Surgery | 160 | 81 | 79 | 49.3 |

We believe that if members of the Legislature understood the reasons for our views in reference to these schools as they are conducted today, they would agree with us. We are just as anxious as anyone that the poor boy should have a chance to study medicine but we are also anxious that the poor boy should not waste his time and money in securing an education which is inadequate to enable him to secure the right to practice his chosen profession. Furthermore in Harvard, Tufts and Boston University, all medical schools regarded as high grade and located in Massachusetts, financial aid to the extent of over $12,000 annually is provided to help the poor boy to study medicine.

We favor the removal of the restriction upon appointment to membership in the Board of Registration, not because we favor or oppose the appointment of a majority of any one school of Medicine but because we believe that the Governor should have a free hand in the appointments and should not be embarrassed as he was recently in making these appointments. At the present time the membership of the Massachusetts Medical Society is somewhat over 4100. The membership of the Massachusetts Homeopathic Medical Society is about 400 and of these a majority are also members of the Massachusetts Medical Society. Of those who remain a certain number are excluded from membership in the Board because of their connection with the faculty of the Boston University School of Medicine. Thus as the law stands today the Governor is practically compelled to select three members of the Board of Registration from among a very few members of the Massachusetts Homeopathic Medical Society. We do not either favor or oppose the appointment of Osteopaths on the Board. As a matter of fact the Secretary of the Board at the present time is an Osteopath and he is elected by the Board itself.

Thus we advocate the removal of the restriction placed upon appointment to membership in the Board of Registration. We advocate provision for the supervision of the equipment and instruction given in our medical schools by some competent body, lay or medical, and we advocate the maintenance of a single standard for the right to practice medicine.

We seek these objects because we believe they are practical, reasonable and just provisions for the public welfare. They are the recommendations of a Committee of the Legislature who studied the problems presented to them from every angle and listened patiently to all parties who wished to be heard. We are sure that any fair minded body of citizens after similar study would come to similar conclusions. We ask you to consider the matter impartially and if you then agree with us we ask your aid in carrying out the recommendations of the Recess Committee in so far as they pertained to the practice of Medicine.

## MISCELLANY

### CONNECTICUT MORTALITY STATISTICS: 1924

WASHINGTON, D. C., October 19, 1925.—The Department of Commerce announces that the 1924 death rate for Connecticut was 1,130 per 100,000 population as compared with 1,201 in 1923. This decrease in 1924 is largely accounted for by decreases in the death rates from pneumonia, all forms (from 116 to 95 per 100,000 population), influenza (from 31 to 15 per 100,000 population), tuberculosis, all forms (from 90 to 82), measles (from 11 to 3), nephritis (from 37 to 82), whooping cough (from 9 to 5), diabetes mellitus (from 23 to 19).

Diseases of the heart and blood vessels showed little change.

An increase in 1924 is shown in the death rate from cancer (from 99 in 1923 to 105 per 100,000 population).

### BEQUEST TO HOSPITALS

UNDER the terms of the will of Mrs. Emma H. Slade, widow of William Gerry Slade, the University of Vermont will receive a trust fund of $50,000 for a memorial to her daughter and one-third of the residuum after death of persons named shall be given to the Massachusetts General Hospital to be used in any way the Trustees of the Hospital wish, provided that a suitable tablet be erected in memory of William Gerry Slade. If this residuum does not amount to $25,000 a sufficient sum shall be taken to make the bequest equal this amount. The remainder of the trust shall be given to the Fordham Home for Incurables.

### THE KIWANIS HEALTH CAMP IN PITTSFIELD, MASSACHUSETTS

THE Director of this Camp, Miss J. D. Sisson, reports that for the season 1925, 116 children (73 boys and 43 girls) were registered and received 1438 days of service. The plan adopted this year was to give a month's service to the girls, between two periods of two weeks each to the boys. This plan demonstrated the advantage of spending a longer continuous time in the camp, for the girls gained from one-half to nine pounds, while the boys gained one-quarter to three and a half pounds. It was noted that in some instances where the boys returned to

the Camp that weight had been lost during the period when they were not in Camp. This demonstration seems to show that two periods of two weeks each in the Camp with an interim is less valuable than a continuous stay of one month.

The total expense for the eight weeks at the Camp was $1389.25. This included salaries. The per capita cost was $11.98. The cost of food was nine cents per child per day. A light lunch was served in the morning on arrival at the Camp, a hearty dinner at noon and a light lunch before returning home at night. The day was divided into rest, play and duty periods.

The health and school departments secured the coöperation of Drs. Monroe and Gannon who conducted a survey of undernourished children. From the children examined selections were made of those needing this care.

The citizens of Pittsfield have taken much interest in this work and have helped by many donations.

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## ENDOWMENT FUND CAMPAIGN, THE PHYSICIANS' HOME, INC., CAMPAIGN HEADQUARTERS

Times Building, 42nd Street & Broadway, New York, N. Y.

ANNOUNCEMENT was made under date of October 14 by President Robert T. Morris, M.D., of The Physicians' Home, Inc., that an endowment campaign has been started by the Directors of the Home for the purpose of raising funds to endow a *national home* for aged and incapacitated physicians who are left without financial resources in the autumn of life.

The sum sought for the home has not yet been determined, but it should run into several millions of dollars, so as to guarantee the upkeep, through interest, of the national home and the several smaller units to be placed in the different states as may be determined later.

The Physicians' Home, Inc., is not an experiment in any sense. Four years ago one unit was established at Caneadea, N. Y., through the generosity of Dr. Stephen V. Mountain, who generously donated the property and building at Caneadea, N. Y., and it has met with such great success that the directors believe it their duty to enlarge the scope of the enterprise, because of the large waiting list which they are unable to accommodate at the Caneadea Unit.

The general plan, outlined by Dr. Robert T. Morris and his associates, is to care for a thousand or more phyiscians at the national home and a dozen or more individuals in the smaller units.

At the present writing it would seem that a million and a half or two million dollars would be necessary, which sum would be invested in bonds of the securest and highest earning value, so as to secure an adequate return in interest to maintain the home and the units without recurring appeals to the medical profession or to the lay-man and woman.

The directors have in mind certain properties that will be had through gift or purchase. The character of the directors is such that the project is guaranteed as to its worthiness and feasibility.

The directors have had the project of a national home in mind for several years, but being practical men they thought they would try it out in the unit established at Caneadea, N. Y., and the success of this unit has been such that they now feel the time is propitious for a national campaign to which the medical profession will be asked to subscribe as their circumstances permit and public spirited citizens also will be aksed to contribute.

The plan of campaign is not in any sense a "Drive." The funds are to be secured through the organization of interested groups in the various cities and towns, and it will take probably a year in this way to put over a campaign that will be dignified and in accordance with the high standard and ethics of the medical profession.

Dr. Charles H. Mayo of Rochester, Minn., has given his unqualified endorsement to the movement and is heading the Committee of Sponsors who will have the campaign in charge. Other prominent physicians and lay-men will also serve as sponsors.

The officers and directors of The Physicians' Home, Inc., are Robert T. Morris, M.D., President, 114 East 54th Street, New York, N. Y.; William H. Dieffenbach, M.D., Vice President, 50 Central Park West, New York, N. Y.; Albert G. Weed, M.D., Treasurer, 152 West 57th Street, New York, N. Y.; Silas F. Hallock, M.D., Secretary, 901 Lexington Avenue, New York, N. Y.; Warren Coleman, M.D., 59 East 54th Street, New York, N. Y.; Max Einhorn, M.D., 20 East 63rd Street, New York, N. Y.; Wolff Freudenthal, M.D., 24 W. 88th Street, New York, N. Y.; J. Richard Kevin, M.D., 252 Gates Avenue, Brooklyn, N. Y.; Stephen V. Mountain, M.D., Olean, N. Y.; Ralph Waldo, M.D., 54 West 71st Street, New York, N. Y.

The campaign will be in charge of Mr. Charles Capehart and Mr. James F. MacGrath, and the national headquarters have already been opened on the 22nd floor of the Times Building at 52nd Street and Broadway, New York City, to which all inquiries should be sent.

All checks should be drawn to the order of "The Physicians' Home, Inc.," and should be forwarded to Dr. Albert G. Weed, National Treasurer, 22nd floor of the Times Building, 42nd Street and Broadway, New York City.

This is the first movement of its kind for physicians in America seeking to secure funds, the income from which will sustain an institution or a series of institutions, having for their pur-

pose the care of those in the medical profession who through generosity, unpaid service, or who through their devotion to the pure science of medicine and laboratory investigation with its small financial return, or who through illness or incapacity find themselves in their declining years unable to provide themselves and their dependents with the necessaries of life.

Of course, the medical profession has its percentage of those who have not had the training or opportunity to lay away sufficient money to finance them in their old age. Then, there are those who have not had the habit of collecting their bills, and who have suffered thereby; and it also will include the younger men in the profession, who, falling ill, have no place to go and none to care for them during their illness. To these latter this home and its unit will prove a great blessing and God-send in administering to their needs until they regain health and can again take up the work of their profession.

This is not intended as a pauperizing movement, nor is the campaign to be one in which there is to be a "sob-element." It is rather to be a dignified effort on the part of the profession itself to take care of its own needy ones and who ask the coöperation of the generous and well-to-do lay-man and woman to help.

From time to time we shall take pleasure in publishing the news of the campaign as it proceeds and it is our earnest hope that the medical profession will answer the call and will send generous contributions to the National Treasurer without waiting to be solicited further.

In no case more than in this appeal can one more definitely give twice by giving what he can quickly. The sooner funds are received the sooner the enterprise will be serving the deserving physicians.

The name tentatively selected for the Home is "Tranquility"; a name that adequately defines peaceful comfort to all found within its walls.

The general plan is to have the Home so laid out that it will typify a real home within which are to be found all those little creature comforts essential to the peace of body and mind of those who are to be the beneficiaries.

One of the features will be a laboratory where the old physician may continue his investigations and study, and thus give him an opportunity of employing head and hand and heart for the advancement of his profession.

Another feature of the Home will be provision for the wife or other dependents of the physician so that families may not be broken up.

It is anticipated that the campaign will be inaugurated by a banquet in New York City to which the profession generally will be invited, as well as prominent lay-men and women.

Speakers of national repute and standing will launch the enterprise.

## EVOLUTION

A SERIES of debates have been held in several cities of the Pacific coast participated in by Dr. Riley, a Christian Fundamentalist, and Dr. Cantrell of the Science League of America. At one of these meetings an audience of about five hundred placed itself on record according to a report in Science (ten to one) that the resolution that the doctrine of evolution is a fallacy and should not be taught in the public schools of America.

R. R. Huster of the University of Oregon sums up his conclusions as follows:

"This mixture of misrepresentation, irrelevance and inconsistency was the case against evolution as propounded by a man advertised as being a prominent fundamentalist. These and similar "arguments" may well bring about, in Oregon and other states, laws which forbid the teaching of evolution in the tax-supported schools. People who sit and applaud such puerilities have a lot to learn. Scientists who find themselves, at such a time, out of touch with and therefore distrusted by the mass of the people, with their capital tied up in a position their tenure of which is subject to the caprice or fears of those in authority, may, in my opnion, take certain lessons to heart also."

## ARE YOU LICENSED TO PRACTICE IN MICHIGAN?

AN inquiry has been received at the JOURNAL Office for information regarding two or more physicians practising in Boston who are also licensed to practice medicine in the State of Michigan.

Should you have these qualifications please communicate with the BOSTON MEDICAL AND SURGICAL JOURNAL.

## DAILY HABITS IN THE FIFTH GRADE

THE American Child Health Association in its recent health survey of 86 cities has gathered the following unique data:—

The average American child of the Fifth Grade

Rises at 6 minutes after 7 in the morning.
Goes to bed at 5 minutes of 9.
Has 10 hours and 11 minutes sleep.
Drinks 1.4 glasses of milk.
Takes a bath once a week.
Usually washes his teeth at least once a day.
Visits the dentist about once in two years.

That twenty-three per cent are vaccinated against smallpox is further information brought out by this survey.

## RECENT DEATHS

**SQUIRE**—DR. ABIRAM F. SQUIRE, a practitioner of Newport, R. I., died at the Newport Hospital October 4, 1925, at the age of 79, of cerebral hemorrhage.

He was born February 26, 1846, in Buffalo, N. Y., and was attending school there at the outbreak of the Civil War. At the age of 16 he enlisted with the Seventy-fourth New York Regiment, serving until after the war. In 1867 he was graduated from the Harvard Medical School. He was one of the founders of the Lawton Warren Post, G. A. R. He was a member of the Rhode Island Medical Society.

GUILD—Dr. Thomas Ezra Guild died at his home in Mattapan, after a brief illness, October 9, 1925, at the age of 57.

He was born in Coventry, Vt., in 1868, took his M.D. at the University of Vermont in 1892, and settled in Mattapan, joining the Massachusetts Medical Society in 1896. He was a Fellow of the American Medical Association and of the Dorchester Physicians' Club, and a member of Rabboni Lodge of Masons. He is survived by his widow, who was Grace Allen of Hyde Park, and by two sons.

## OBITUARY

### MASSACHUSETTS ASSOCIATION OF BOARDS OF HEALTH

#### Resolutions

Whereas:—By the death of Dr. Eugene R. Kelley, Commissioner of Health of the Commonwealth of Massachusetts, the Massachusetts Association of Boards of Health has lost an active member and a wise councilor:

Resolved:—That the Association records its appreciation of Dr. Kelley's services in the promotion of public health in this Commonwealth and its regret at the loss which it has sustained.

Resolved:—That the Association expresses its sympathy to the bereaved family.

Resolved:—That these Resolutions be spread upon the records of the Association, and the Secretary be directed to send a copy to the family.

Signed,
M. Victor Safford,
Francis Geo. Curtis.

## CORRESPONDENCE

### THE LATE DR. DAVID W. CHEEVER'S LECTURES ON SURGERY

*Mr. Editor:*

Those who had the pleasure and profit of listening to Dr. Cheever's lectures on surgery have never forgotten them, and at the present time, when such charm of diction is so rare, it is a pleasure to recall them. Some of us also remember Dr. Cheever's illuminating articles in this Journal on "Medicine as a Trade" and "Medicine as a Profession."

Let us for a moment try to visualize the lecturer, and attempt to describe the charm of the lecture, though this is impossible to do adequately.

The lecture hall was always full at this hour.

Dr. Cheever would enter quickly, at exactly the appointed time, and, stepping to the center of the room, take from his pocket a small card, on which were all the notes he found it necessary to arm himself with. He was invariably clad in gray, immaculate, usually wearing a cutaway suit and gray tie to match. When he began his lecture he always brought his hands together in front of the chest, the fingers lightly touching; he would then begin, saying (perhaps), "Gentlemen, at the last lecture I was considering fractures of the upper end of the humerus, and we will proceed with the subject."

Whatever Dr. Cheever was speaking of, there was an hour in which one could have heard a pin drop.

The style, simple, as with all great men, the words carefully chosen, without effort, and the voice soft and agreeable, yet perfectly heard. These rare attributes of the lecturer, combined with Dr. Cheever's great knowledge of his subject, formed a happy combination rarely seen at this day, and to many of his audience the time of the lecture seemed a few minutes rather than an hour.

Very truly yours,
Wm. Pearce Coues, M.D.

Brookline, October 10, 1925.

### POLYORCHIDS

219 Foch Street, Ellwood City, Pa.,
October 13, 1925.

*Editor, Boston Medical and Surgical Journal:*

Within the past few days I have examined two *polyorchids*. The population here is not quite 10,000, so I thought my find unusual enough to report (although Gould and Pyle's "Anomalies and Curiosities of Medicine" mentions a . . . sextorchid!).

Case 1. A Pole. Aged 40. Married and has three normal children. The patient, however, is slowly going blind from Retinitis Pigmentosa. Pt. claims that his father and his own brothers were normal in every way. . . . The testicles lie in the bottom of the scrotum.

Case 2. An American. Aged 30. Married and has two normal children. Wife of pt. has often boasted to her neighbors of her frequent abortions. Pt. himself seems normal otherwise and occupies a good social standing; however, he does most of his wife's housework when he gets home from his own work. . . . His testicles, like the foregoing case, lie at the bottom of the scrotum.

Another doctor, here, tells me that he saw a triorchid some time ago, not far from this town.

Very truly yours.
L. W. Darrah, M.D.

### RESUME OF COMMUNICABLE DISEASES REPORTED IN MASSACHUSETTS

#### September, 1925

##### General Prevalence

The common communicable diseases showing an increase over last month were chickenpox, diphtheria, scarlet fever and whooping cough:

| | Sept., 1925 | Aug., 1925 | Sept., 1924 |
|---|---|---|---|
| Chickenpox | 83 | 62 | 66 |
| Diphtheria | 288 | 200 | 318 |
| Scarlet fever | 225 | 179 | 367 |
| Whooping cough | 723 | 600 | 222 |

##### Rare Diseases

*Anterior poliomyelitis* was reported from Adams, 2; Attleboro, 1; Barnstable, 1; Beverly, 1; Boston, 10; Brookline, 1; Cambridge, 1; Colrain, 1; Danvers, 1; Fairhaven, 1; Fall River, 1; Great Barrington, 1; Lawrence, 1; Lynn, 1; Malden, 1; Melrose, 1; Newton, 2; North Adams, 1; Peabody, 1; Pittsfield, 1; Plainville, 1; Revere, 1; Somerville, 1; Taunton, 2; Waltham, 3; Watertown, 1; Westport, 1; Winchester, 2; Winthrop, 1; total, 44.

*Dog-bite requiring anti-rabic treatment* was reported from Billerica, 1; Boston, 1; Lowell, 9; Medfield, 1; Revere, 1; Winthrop, 2; total, 15.

*Encephalitis lethargica* was reported from Boston,

3; Fall River, 1; Haverhill, 1; Lynn, 2; Malden, 1; Methuen, 1; Quincy, 1; Somerset, 1; Springfield, 1; Taunton, 1; Worcester, 1; total, 14.

*Epidemic cerebrospinal meningitis* was reported from Boston, 2; Hingham, 1; Lawrence, 1; Lowell, 1; Malden, 1; Medford, 2; Northampton, 1; Plainville, 1; Rehoboth, 1; Worcester, 1; total, 12.

*Pellagra* was reported from Boston, 1.

*Rabies* was reported from Holyoke, 1.

*Septic sore throat* was reported from Boston, 3; Cambridge, 1; Framingham, 1; Haverhill, 1; Lawrence, 1; total, 7.

*Smallpox* was reported from Medford, 1.

*Tetanus* was reported from Boston, 3; Quincy, 2; total, 5.

*Trachoma* was reported from Cambridge, 2; Watertown, 1; total, 3.

DISTRIBUTION

*All Communicable Diseases*

| | Sept., 1925 | Sept., 1924 |
|---|---|---|
| Total cases (all causes) | 3,266 | 2,851 |
| Case rate per 100,000 population | 80.9 | 71.2 |

*Certain Prevalent Diseases*

| *Diphtheria* | Sept., 1925 | Sept., 1924 |
|---|---|---|
| Total cases | 288 | 318 |
| Case rate per 100,000 population | 7.1 | 7.9 |

Cities and towns noticeably exceeding their median endemic indexes*:

Worcester (26) 59

| *Measles* | Sept., 1925 | Sept., 1924 |
|---|---|---|
| Total cases | 269 | 143 |
| Case rate per 100,000 population | 6.7 | 3.6 |

Cities and towns noticeably exceeding their median endemic indexes*:

Fall River (1) 24 Worcester (4) 110
Lowell (5) 41

| *Scarlet Fever* | Sept., 1925 | Sept., 1924 |
|---|---|---|
| Total cases | 225 | 367 |
| Case rate per 100,000 population | 5.6 | 9.2 |

| *Tuberculosis, Pulmonary* | Sept., 1925 | Sept., 1924 |
|---|---|---|
| Total cases | 435 | 436 |
| Case rate per 100,000 population | 10.8 | 10.9 |

| *Tuberculosis, Other Forms* | Sept., 1925 | Sept., 1924 |
|---|---|---|
| Total cases | 42 | 61 |
| Case rate per 100,000 population | 1.0 | 1.5 |

| *Typhoid Fever* | Sept., 1925 | Sept., 1924 |
|---|---|---|
| Total cases | 80 | 80 |
| Case rate per 100,000 population | 2.0 | 2.0 |

| *Whooping Cough* | Sept., 1925 | Sept., 1924 |
|---|---|---|
| Total cases | 723 | 222 |
| Case rate per 100,000 population | 17.9 | 5.5 |

Cities and towns noticeably exceeding their median endemic indexes*:

| | | | | | |
|---|---|---|---|---|---|
| Falmouth | (0) | 5 | Norwood | (0) | 8 |
| New Bedford | (1) | 24 | Melrose | (0) | 9 |
| Avon | (0) | 30 | Swampscott | (0) | 19 |
| Brockton | (4) | 29 | Belmont | (1) | 11 |
| Cambridge | (24) | 39 | Worcester | (10) | 73 |
| Newton | (4) | 33 | Greenfield | (0) | 15 |

*The median endemic index is obtained by arranging in arithmetical sequence the monthly totals of reported cases for the past five years and selecting the middle figure. The numbers in parentheses after the name of each city and town indicate the median endemic index for that city or town; the numbers without parentheses indicate the cases reported during the current month.

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CONNECTICUT DEPARTMENT OF HEALTH

MORBIDITY REPORT FOR THE WEEK ENDING OCTOBER 3, 1925

| | | | |
|---|---|---|---|
| Diphtheria | 18 | Chickenpox | 2 |
| Last week | 14 | German measles | 1 |
| Diphtheria bacilli carriers | 4 | Influenza | 3 |
| Scarlet fever | 27 | Pneumonia, lobar | 15 |
| Last week | 15 | Poliomyelitis | 2 |
| Typhoid fever | 13 | Septic sore throat | 1 |
| Last week | 11 | Trichinosis | 2 |
| Measles | 22 | Tuberculosis, pulmonary | 21 |
| Last week | 8 | Tuberculosis, other forms | 2 |
| Whooping cough | 69 | Chancroid | 1 |
| Last week | 44 | Gonorrhea | 32 |
| Bronchopneumonia | 11 | Syphilis | 30 |
| Cerebrospinal meningitis | 2 | | |

## REPORTS AND NOTICES OF MEETINGS

LAWRENCE GENERAL HOSPITAL Clinical Staff Meeting was held Tuesday, October 13, 1925.

The subject was the reading in extenso of the minutes of a clinical meeting of the staffs of two Brooklyn, N. Y., Hospitals, consisting of abstracts of records of cases ending in deaths, criticisms of treatment and diagnosis and the records of the cases.

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### CENSORS' MEETING

THE censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, November 5th, 1925, at 4.00 o'clock.

Candidates should make personal application to the Acting Secretary, and present their medical diploma at least one week before the examination.

RICHARD H. MILLER,
*Acting Secretary.*

270 Commonwealth Avenue.

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### THE HARVARD MEDICAL SOCIETY

THE next regular meeting of the Harvard Medical Society will be held as usual in the amphitheatre of the Peter Bent Brigham Hospital, Oct. 27, 1925, at 8.15 p. m. The program follows:—

1. Demonstration of Cases.

2. Direct Observation on the Heart—Dr. C. K. Drinker.

All members of the Medical Profession, Medical Students and Nurses are invited.

## THE NORFOLK DISTRICT MEDICAL SOCIETY

A STATED meeting of the Norfolk District Medical Society will be held at the Wrentham State School—Tel. Wrentham 24, October 27th, 1925.

Dr. George L. Wallace, leading authority in the care and training of mentally defective children, and in institutional management has again favored us in arranging another interesting program.

We hope this meeting will be largely attended as those of previous years.

PROGRAM

4 P. M., school visit. 5 P. M., business meeting, followed by presentation of cases from the clinic of Dr. George L. Wallace. Collation. Entertainment. Please notify the secretary in writing of changes in address. Next meeting of Censors, November 5th, 1925.

FRANK S. CRUICKSHANK, *Secretary.*

23 Bay State Road, Boston.

## SUFFOLK DISTRICT MEDICAL SOCIETY

OCTOBER 29, *Stated Meeting* (Changed from October 28th)—Election of Secretary to fill vacancy caused by the death of Dr. Spooner. The Nominating Committee presents the name of Dr. Arthur H. Crosbie. The election of one member of the Nominating Committee and one member of the Auditing Committee. Papers: "The Practical Significance of Jaundice." Dr. Chester M. Jones. "An Easy Method of Estimating the Amount of Jaundice by means of the Blood Serum." Dr. William P. Murphy.

NOVEMBER 18, *Surgical Section*—"Industrial Surgery." By Dr. W. S. O'Neill Sherman of the Bethlehem Steel Works. Discussion opened by Dr. Irving Clark of the Norton Co., Worcester, Mass. Further notice later.

JANUARY 6, *Medical Section Meeting*—Meeting postponed from December in order to procure Dr. W. J. MacDonald of Toronto who will speak on "Experimental work in High Blood Pressure."

JANUARY 27, *Combined Meeting with Boston Medical Library*—"Medical Experience of an Explorer." Dr. J. Hamilton Rice.

FEBRUARY 24, *Surgical Section Meeting*—"Post-operative Care of Surgical Cases." Dr. Dean Lewis—Chicago. "Surgical Convalescence." By Dr. John Bryant.

MARCH 31, *Medical Section Meeting*—Subject to be announced later.

APRIL 28, *Annual Meeting, Election of Officers*—"Some Diagnostic, Prognostic and Therapeutic Aspects of Disorders of the Blood." Drs. George R. Minot, Cyrus C. Sturgis, Raphael Isaacs of the Collis P. Huntington Hospital of Harvard University.

The Medical Profession are cordially invited to attend all these meetings.

C. MORTON SMITH, M.D., *President, Suffolk District Medical Society.*
RICHARD H. MILLER, M.D., *Secretary Protem, Suffolk District Med. Soc.*
ELLIOTT P. JOSLIN, M.D., *Chairman, Medical Section, Suffolk District Medical Society.*
ROBERT B. OSGOOD, M.D., *Chairman, Surgical Section, Suffolk District Medical Society.*
MALCOLM STORER, M.D., *Boston Medical Library.*

## ESSEX SOUTH DISTRICT MEDICAL SOCIETY

NEXT meeting Wednesday, November 4, at the Salem Hospital.

5 P. M.—Clinic by members of the Staff.

7 P. M.—Dinner.

Speaker, Francis B. Ellis, M.D., who will speak on Medical Experiences in India.

Dr. Ellis is on a three months leave from The Indian Medical Service, where he has had most extensive and interesting experiences. We think we are most fortunate in procuring Dr. Ellis to speak to us, and hope there will be a large attendance.

CENSORS MEETING

The Censors of the Essex South District Medical Society will meet at the Salem Hospital, Thursday, November 5, at 3.30 P. M.

Candidates for admission to the Society should, at once, present their applications and diplomas to the Secretary.

R. E. STONE, M.D., *Secy.*

## THE WORCESTER DISTRICT MEDICAL SOCIETY

THE October meeting of the Worcester District was held at the Grafton State Hospital on Wednesday, October 14, 1925. At 4.30 in the afternoon, Dr. Harlan Paine, Superintendent of the Hospital, conducted an inspection of the Hospital for the visiting members. At 5.30 Dr. Royal P. Watkins presided at a short business meeting. Resolutions on the recent deaths of Dr. James Taylor and Dr. Michael Halloran of Worcester and Dr. Edmund Douglass of South Barre were read and accepted. Dr. Arthur W. Marsh as chairman of the Publicity Committee, read the report of this committee for the past year. Dr. Watkins called to the attention of the members that special blanks must be used by physicians in this state in writ-

ing prescriptions for hypodermic syringes and needles. These blanks are furnished by the State Board of Registration upon application.

At six o'clock a bountiful repast including freshly made cider was enjoyed by the Society. This Hospital always provides fine food and plenty of it, thanks to Dr. Paine.

After supper Dr. Watkins introduced Dr. Allen Rowe of Boston who gave a most interesting talk on "Differential Diagnosis of Endocrine Diseases from an Objective Standpoint." This subject was extremely hard to present but Dr. Rowe proved beyond doubt that he knew this subject thoroughly and he is to be complimented on the way that he delivered his talk to the Society. His conclusions were that we knew the most about the pituitary gland and that the only other glands we knew much about were the thyroid, the adrenals, the ovaries and the testicles. We can only make a diagnosis of endocrine disorders after a careful and thorough examination including all the laboratory tests.

The meeting was adjourned at nine o'clock.

The next meeting of this Society will be at the Worcester State Hospital on Wednesday, November 11, 1925.

EARL E. FIPPHEN.

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## MEETING OF THE HARVARD MEDICAL SOCIETY

THE Harvard Medical Society met at the Peter Bent Brigham Hospital on the evening of October 13th. Dr. Christian presided.

Two cases were presented. The first was one of aortic aneurysm in a man of forty-four years. There was a history of syphilitic infection and during the past ten years of pain in the chest. Two years ago an expansile mass was found in the third interspace. An operation was performed to insert a wire into the aneurysm. This gave relief from pain for a year. Now the patient has returned complaining of a distressing cough and dyspnoea. After study of the case with X-ray it has been decided to wire the aneurysm again with a finer wire to fill in the spaces between the coils of the larger one. With the production of more clot, the pressure causing the cough and dyspnoea may disappear. In the meantime the patient is being given potassium iodide and symptomatic treatment for the cough.

The second case was a man of fifty-seven years, suffering from myxoedema. His complaint was pain in the chest and shortness of breath. Physical examination showed the heart to be enlarged, the skin dry and scaly, body hair scanty; speech slow, blood pressure and metabolism below normal. Under administration of thyroid the diameter of the heart decreased and the metabolism became higher. The old symptoms returned as soon as patient went to work. He is now back in the hospital for further treatment. The great problem in this case is to regulate the metabolic rate, by means of thyroid, at the level which will be optimum for his circulatory and myxoedemic disturbances. Following the demonstration of cases, Dr. D. J. MacPherson addressed the society on Encephalitis Lethargica from a clinical standpoint. As a clinical entity, this disease has come into being during the last decade. It is decidedly a winter disease reaching its highest incidence during the months of November, December and January. There is a wide variation in the age of cases, but the greater number occur in early adult life. The height of an epidemic has often occurred with a wave of influenza, but the possibility of any connection between the two is as yet quite undecided. The age incidence and the seasonal variation are against the virus of poliomyelitis as a cause.

The symptoms may be divided into two groups, general and neurological.

Under general symptomatology, it may be described as a toxic infection. A slight fever appears early in the onset. It is very variable and never lasts over a week. Only thirty per cent of the cases show this fever. The onset of the disease is insidious, so that the fever may have come and gone in many cases before the diagnosis was made.

Other general symptoms are headache, vomiting, hiccoughs, severe constipation and severe erythematous rash. Any or all of these may be present in a given case.

Under neurological symptoms, lethargy may be placed as the most constant finding. When awakened from this unconscious state by shaking, the patient is intelligent, but immediately lapses back into the lethargy. If the patient cannot be aroused, it is usually a serious prognostic sign. Sometimes instead of lethargy, there is an extreme degree of talkativeness and irritability. Often there is marked insomnia. Convulsions or true manic excitement is very rare except in severe cases, Neurasthenic symptoms form one of the difficult sequellae of this disease.

Eye symptoms are quite frequent. The pupils may be unequal and there may be an Argyle-Robertson pupil in one or both eyes. Paralysis of accommodation may also occur. There may be any degree of paralysis of the ocular muscles. A double ptosis is common. External opthalmoplegia, affecting chiefly the up and down movements may be found. Opthalmoscopic examination rarely shows any change in the nerve head.

Many variations occur in the rest of the cranial nerves. There is often a facial paralysis which is sometimes bilateral. Occasionally there are bulbar symptoms. When the basal ganglia are affected, the patient has the weakness, rigidity and slowness of motion, charac-

teristic of the Parkinsonian syndrome. This is the most intractable of all the sequellae. In some cases there are involuntary movements of all types, in others a flaccid paralysis that may involve the sphincters. Incontinence is usually due to the lethargy, however, rather than to an organic lesion.

The prognosis in this disease is doubtful and rather poor. Twenty-five to thirty per cent get well. About the same number die. The remainder are left with some difficulty that carries on upwards of a period of years. In children there are more apt to be character defects and mental changes.

Dr. Hans Zinsser spoke on "Experimental Aspects of the Encephalitis Problem." The burning question in recent research on this disease has been the possible relationship between the etiological factor and other viruses, especially the virus of Herpes Simplex. The virus of Herpes Simplex may be easily inoculated into the cornea of rabbits. It has thus been transmitted from rabbit to rabbit through many generations. The mortality of the rabbits varies from ten to one hundred per cent depending on the virulence of the particular strain of virus used. With brain inoculations the rabbits always die.

Herpes is a very insignificant thing in humans. It is regarded by Italian investigators as a general infection; the vesicle being only a manifestation of the infection. The virus of Herpes has been found in the spinal fluid of many patients and also in the blood stream in a few cases. It seems to remain in the spinal fluid for months after the attack. Injections of the virus from rabbits into humans intraspinally or otherwise produced no marked symptoms.

Rabbits that receive skin inoculations become rapidly immune to the virus so that they may withstand brain inoculations. If a rabbit survives a severe infection it again becomes susceptible in four or five months.

The evidence regarding passive immunization is rather contradictory. Some investigators have reported successful attempts to immunize with the serum of immune animals. Others have failed to do so. The virus cannot be neutralized by serum in vitro according to most observations, although Flexner has recently reported neutralization by this method. With nearly all filtrable viruses, immunization can only be accomplished by using some form of the living virus. This is because their action is intracellular and they must get inside to cause immunization. A dead virus can't get inside. An attenuated virus must be used as in rabies.

Partially immunized rabbits live longer than controls that are fully susceptible. After a number of days, instead of exhibiting the typical symptoms of herpetic encephalitis, they go into a condition resembling encephalitis lethargica in humans.

There has been almost complete failure in attempts to transmit the virus to rabbits from cases of encephalitis. There were many early reports of success, in which nasal and throat washings were used, but these may be disregarded as representing a form of Herpes. Levaditi and others have with difficulty succeeded in getting passage strains from the central nervous system of patients. The result in rabbits has been practically identical with that due to inoculation with the Herpes virus. It is possible that the virus may have been an incidental finding in these cases of encephalitis in view of the fact that many patients who have never had manifestations of Herpes or encephalitis, are found to have Herpes virus in their spinal fluid.

An English investigator has recently thrown new light on the problem. Instead of injecting the virus at once which he obtained from human cases of encephalitis, he preserved it in glycerine for three or four weeks and then injected it into rabbits. He obtained positive results and was able to transmit the virus in passage and show complete identity with Herpes. This work needs to be confirmed by further experiments.

The fact that the majority of poliomyelitic cases do not show typical clinical pictures makes a similar thing conceivable concerning the Herpes virus. If there are fluctuations of virulence in this virus, it is reasonable that we might be carriers after a mild attack. Such fluctuations might also explain the difference in the effect of the virus on man and rabbits. Marked changes in virulence are common in filtrable viruses when passed from animal to animal. In the case of encephalitis, there may be something in fresh human material which prevents immediate transmission to rabbits. In view of the experimental evidence, obtained thus far, it does not seem safe to abandon any idea of relationship between the virus of Herpes and that of encephalitis.

The meetings of the Harvard Medical Society will be held on the second and fourth Tuesdays of each month throughout the fall, winter and spring months. The main item on the program of the meeting scheduled for Oct. 27th will be an address by Dr. C. K. Drinker on "Direct Observations on the Heart."

## SOCIETY MEETINGS

### District Medical Societies

*Essex North District Medical Society*

Wednesday, January 6, 1926—The semi-annual meeting at Haverhill.

Wednesday, May 5, 1926—The annual meeting at Lawrence.

### New England State Medical Societies

The annual meetings of the New England State Medical Societies are scheduled as follows:

Vermont State Medical Society—St. Johnsbury, Oct. 15-16, 1925.

*Notices of meetings* must reach the JOURNAL office on the Friday preceding the date of issue in which they are to appear.